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A DENTAL SURVEY OF THE ABORIGINES AT HAAST'S BLUFF, CENTRAL AUSTRALIA.[1]

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THERE are a number of records of the teeth of Australian aborigines, the most recent being those by Campbell and Barrett (1953), Barrett (1953, 1957) and Cran (1955, 1957) on the Ngalia (Wailbrai) tribe at Yuendumu. This study is concerned mainly with a different people who were encountered on an expedition led by Professor A. A. Abbie to Haast's Bluff, Central Australia, in August, 1956. The present paper deals with observations on the living natives. A second will describe the casts obtained on that occasion.

MATERIALS AND METHODS.

The natives, previously numbered with the prefix "Y", were examined as thoroughly as conditions allowed. The ages were assessed on any records available, physical development, sex and whatever additional information could be obtained.

At a complete examination of the mouth and teeth with mirror and probe, the following observations were made: (*a*) teeth present; (*b*) caries; (*c*) calculus—arbitrary classification into four degrees; (*d*) attrition—according to the Broca classification: (i) enamel showing slight wear, but no exposure of dentine, or cusp obliteration, (ii) dentine exposed, cusps worn down, (iii) elimination of grooves and occlusal surface features, (iv) crown of tooth almost completely worn down to neck region; (*e*) occlusion—normal, class I, class II (divisions I and II) and class III, Angle's classification being used: (*f*) hypoplasia—nil, mild, severe; (*g*) hypocalcification—nil, mild, medium, severe; (*h*) pigmentation (these grades are based upon the extent of pigmentation as recorded in colour films): (i) gingivæ—grades 1, 2, 3, 4, (ii) mucosa—grades 1, 2, 3, 4, (iii) palate—grades 1, 2, 3, 4; (*i*) depth of gingival crevice in millimetres; (*j*) presence of premaxillary diastemata; (*k*) any abnormalities, variants or pathological conditions, etc.

Lateral X-ray films were taken on 64 of the 145 natives examined. In general the radiological examinations were restricted to those aged between six and 23 years, otherwise they were taken only for definite reasons, such as unerupted teeth, etc. From the lateral X-ray films the following observations were made: (i) state of the dentition; (ii) which apices had closed; (iii) the position of the mental foramen; (iv) condition of the alveolar bone; (v) any abnormalities or variants.

[1] The data upon which this paper is based were collected during a University of Adelaide expedition, financed mainly by the Wenner-Gren Foundation for Anthropological Research, New York, and the University of Adelaide Medical Research Fund.

A total of 115 casts were made, impressions being taken in perforated trays, and built up with "Plasticine", with the use of "Verex", an alginate hydrocolloid. These were cast in green stone.

Photographs of any abnormality were also taken, and an attempt was made to classify pigmentation by the use of colour film.

Age Grouping.

For the purposes of this paper, the following five age groups were adopted:

Group I: infant, showing deciduous dentition only.

Group II: young; first permanent molar erupted, second permanent molar unerupted or partially erupted.

Group III: adolescent; second permanent molar erupted, third unerupted or partially erupted.

Group IV: adult, permanent dentition complete.

Group V: aged; dentition and gingival appearance suggesting age, approximately over 40 years.

By civilized standards this last age grouping may appear to commence relatively early; but in general by about 40 years the natives did, in my view, appear to be in a definitely different group from those under that age. They presented an aged appearance with grey hair, Broca (iii) to (iv) attrition and a less vigorous physical condition. They were broadly accepted as being the elders of the tribe.

Environment of the Natives Examined.

One hundred and forty-five natives were examined, the majority belonging to the Pintubi or Aranda tribes. Other tribes well represented were the Ngalia, Kukatja and Pitjandjara. No attempt has been made to distinguish between the tribes in respect of dental findings.

The majority lived under native conditions in camps around the station—i.e., within a five-mile radius—while others lived on the station in galvanized iron buildings. All were given a Government ration, the size of which depended largely on age. However, the relationship between the ration issued and ration actually consumed by each individual cannot be estimated, because the family method of distribution varied.

The ration included flour, baking-powder, sugar, tea, tinned and/or dehydrated vegetables, tinned fruit juice, meat, rice and dried fruit. The mission store sold tinned foodstuffs, sweets, biscuits and cool drinks, which children in the vicinity of the camp quite often procured. This diet was supplemented by their natural food (see Campbell, 1928, 1938, 1939)—i.e., seeds, honey, nuts, plants, etc.—which they gathered and the meat which they hunted.

The food intake thus comprised both the basic native diet, which was rich in protein and vitamins and relatively poor in fats, and the rations, which had a moderate content of protein and vitamins *C* and *B* and a high content of carbohydrates.

Water.

Water samples were collected from the following four sources:

1. Haast's Bluff Bore. This was used by natives, but not by whites. The total salt content amounted to 1350 parts per million, chloride accounting for 215 p.p.m. and fluorine for 1·8 p.p.m.

Previous estimations by M. J. Barrett in 1953 and 1954 (personal communication) showed the total salt content to vary between 1200 and 1632 p.p.m. and the fluorine content between 2·3 and 2·7 p.p.m. A Department of Animal Industry estimate, of unknown date, disclosed a salt content of 4332 p.p.m. and a fluorine content of 2·2 p.p.m. Thus the total fluorine intake from this source amounts on an average to about 2·3 p.p.m.

2. Papunja Bore. This was at a new settlement being established to replace Haast's Bluff. This water had a total salt content of 840 p.p.m. and a fluorine content of 0·9 p.p.m.

3. Ulambarra Spring. This was a water hole approximately 23 miles north-east of Haast's Bluff. It was used only by wandering natives, and had a total salt content of 160 p.p.m. and a fluorine content of 0·3 p.p.m.

4. Tuliputa Spring. This was at the entrance to Tuliputa Gorge, approximately 50 miles west of Haast's Bluff. It was used only by wandering natives, not being a regular source of water. It had a total salt content of 175 p.p.m. and a fluorine content of 0·4 p.p.m.

Findings.

Dental Caries.

Dental caries will be discussed under the headings of incidence, defective and missing teeth, sex differences, distribution patterns and type of caries.

Incidence.

The incidence of caries in the number of subjects examined, the average number of carious teeth per subject, and the number of deciduous and permanent teeth examined are shown in Table I. This does not include missing teeth, the loss of many being most certainly attributable to caries.

TABLE I.
Incidence of Caries.

Material Examined.	Number Examined.	Number Showing Caries.	Percentage Showing Caries.
Aborigines	140	89	63·6
Teeth:			
Deciduous	478	31	6·5
Permanent	3201	336	10·5
Total	3679	367	10·0
Average number of carious teeth per subject	2·6	—	—

Table II shows the percentage incidence of caries by age groups, with no distinction between permanent and deciduous dentitions.

TABLE II.
Subjects with Carious Teeth by Age Groups.

Group.	Number of Subjects.	Number with Caries.	Percentage with Caries.
I	19	1	5·3
II	15	9	60·0
III	19	12	63·2
IV	54	43	79·8
V	33	22	66·7

The mean incidence and occurrence of caries in deciduous and permanent teeth by age groups is shown in Table III.

The pattern of increased caries incidence with age groups is significant. Of the children in Group I, only one child exhibited caries, and then in only one tooth; this gave a very small percentage of caries incidence for the group—5·3 of subject incidence and 0·4 of tooth incidence. There followed a rapid increase by the mixed dentition stage, when 15·6% of the deciduous teeth and 4·4% of the permanent teeth were carious, the subject incidence increasing enormously to 60%.

From the mixed dentition stage on, the subject incidence remained relatively constant, although the tooth incidence rose slowly in Group III to 5·4%, then rapidly to 11·5% and 12·6% in Groups IV and V respectively.

Defective and Missing Teeth.

Estimations of the defective-missing (D–M) values present in general a more satisfactory means of assessing

the state of the dentitions in relation to caries than the figures for caries alone.

Table IV gives the mean number of missing permanent teeth per subject by age groups. Two means are given: (i) including dental mutilations; (ii) excluding dental mutilations.

Table V shows the mean D–M value per subject by age groups. As above, two values (i.e., including and excluding dental mutilations) have been given. The latter must be considered as the actual D–M value in relation to the occurrence of caries, since the mutilations are usually performed on virgin teeth. Figure I shows diagrammatically the D–M pattern by age groups.

Sex and age-group differences in D–M values are shown in Table VI.

In the natives examined, the two figures which may be given as an indication of dental decay are (i) the incidence

TABLE III.

Percentage of Teeth Showing Caries by Age Groups.

Group.	Number of Subjects.	Number of Teeth.	Number of Carious Teeth.	Percentage of Carious Teeth.	Mean Number of Carious Teeth per Subject.
I	19	286	1	0·4	0·05
II	15	192 deciduous	30	15·6	2·0 deciduous
		168 permanent	8	4·4	0·53 permanent
III	19	535	29	5·4	1·53
IV	54	1665	192	11·5	3·2
V	33	833	107	12·6	3·24

of caries which was 63·6%, or (ii) the D–M value, which was 72·9%, not due to dental mutilation but, presumably, to decay. Eight cases of advanced periodontal disease were noted. In each of these cases either caries was present or there was evidence that the tooth loss had been due, in part at least, to caries. The D–M value is of greater significance for obvious reasons.

Of a total of 3679 teeth (478 deciduous and 3201 permanent), 367 showed caries and 668 were defective or missing (excluding mutilations). The mean number of carious teeth was 2·6, 10% of the teeth showing caries; but the mean D-M value was 4·7, 16·9% of the teeth being defective or missing. These figures alone indicate the vastly different results obtained by using the two different indices.

The D–M values for Groups I and II are the same as the caries incidence values; while in Group III, as only two permanent teeth were missing in the 535 examined, the two results differ but slightly. After attainment of the complete dentition (i.e., Group IV), a D–M value of 4·46 (excluding mutilations) was obtained. This rose to 10·03 in Group V. Thus the over-all D-M value for permanent teeth was 5·26.

As with all similar estimations, the over-all figure for the entire survey is rather misleading, as it is so dependent upon the numbers in each group. For example, a survey covering mainly children will show a low incidence of caries, while if the majority of subjects examined are adult (e.g., the present survey) the incidence of caries will be correspondingly higher.

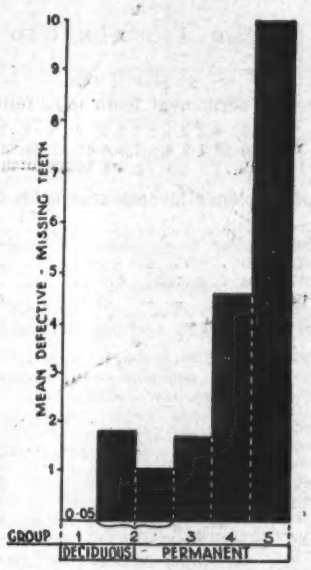

FIGURE I.

Diagram to illustrate the mean incidence of defective-missing teeth in the various age groups.

The caries pattern throughout the life of average males in this group may be estimated thus. Relative immunity is present till mixed dentition, at which stage there appears a rapid increase in caries in the deciduous dentition. (The rapid caries increase evident in Europeans during adolescence does not occur in the average native at the same stage, but becomes manifest later.) In the permanent dentition during adolescence there is at least one tooth exhibiting caries. On completion of the adult dentition, and with increased food stagnation areas, the caries rate rises until the average adult will exhibit three carious teeth and two teeth missing—one owing to caries, and the other to dental mutilation. In an average old man's dentition there are three carious teeth and seven teeth missing, mutilation accounting for at least one of the latter.

Sex Differences.

With the exception of Group III, females generally showed a slightly greater D–M value, and most markedly in

TABLE IV.

Mean Number of Missing Permanent Teeth per Subject by Age Groups.

Group.	Number of Subjects.	Teeth Standing.	Roots Present.	Teeth Unerupted.	Teeth Missing.	Teeth Missing from Mutilations.	Mean Number Missing per Subject.	
							Including Mutilations.	Excluding Mutilations.
II	15	192	—	288	—	—	—	—
III	19	535	—	71	2	—	0·11	—
IV	54	1665	—	10	53	12	0·98	0·76
V	33	833	1	369	223	25	6·76	6·0
Total	141	3225	1	—	278	37	1·97	1·71

Group V, in which the D–M values were 17·7 as compared with 7·74 for males. Such a difference in D–M value in the older female natives is difficult to explain. Campbell (1925) has attributed this to coarser diet; but other factors—e.g., calcium loss through menstruation—could also be invoked.

Caries Distribution Patterns.

The incidence of caries in individual deciduous teeth is as follows:

Right	E	D	C	B	A	:	A	B	C	D	E	Left
Upper	3	–	–	–	–	:	–	–	1	1	5	Upper
Lower	6	2	–	–	–	:	–	–	–	4	5	Lower

The incidence in permanent teeth is as follows:

Right	8	7	6	5	4	3	2	1	:	1	2	3	4	5	6	7	8	Left
Upper	19	25	34	13	12	7	9	6	:	7	8	6	13	11	36	21	17	Upper
Lower	25	39	53	18	12	7	1	3	:	2	2	4	14	19	50	39	23	Lower

The order of frequency in deciduous teeth is as follows:

Lower second molar	11
Upper second molar	8
Lower first molar	6
Upper first molar	1
Upper canine	1

The order of frequency in permanent teeth is as follows:

Lower first molar	103
Lower second molar	78
Upper first molar	70
Lower third molar	48
Upper second molar	46
Lower second premolar	37
Upper third molar	36
Lower first premolar	26
Upper second premolar	26
Upper first premolar	24
Upper lateral incisors	17
Upper canines	13
Upper central incisors	13
Lower canines	11
Lower central incisors	5
Lower lateral incisors	3

The surfaces and site affected in deciduous and permanent dentitions are shown in Tables VII and VIII.

The left and right sides of the mandibular and maxillary dentitions yielded 272 and 284 carious teeth respectively. Thus the two sides of both may be said to have about the same caries incidence. The ratio of mandibular to maxillary caries for both deciduous and permanent dentitions was 3:2. The lower second molar was the most commonly involved in the deciduous dentition. The lower first molar was the most frequently affected permanent tooth, representing approximately one fifth of the total D–M permanent teeth.

From Tables VII and VIII, it may be seen that occlusal surfaces were most affected by caries. The most frequent kind was of the smooth surface variety. In the permanent dentition, interproximal caries had by far the highest incidence, representing 50% of the total. Smooth surfaces again were the commonest initiating site, while pit cavities were the next affected. Since occlusal attrition continues in the natives at quite a considerable rate, fissures are readily eliminated, hence their very low incidence in the permanent dentition. Buccal pits on the lower molars were frequently affected by decay.

Type of Caries.

In general the caries, when it occurred, was of the very rapid variety. It seemed, especially with interproximal caries, that once the enamel had been penetrated, the resistance of the remaining tooth structure to the onslaught of decay was greatly lowered, and this led to rapid pulpal involvement. Slow caries was mainly observed on the occlusal surfaces, where attrition continually reduced both the susceptible areas and the actual carious structure.

Dental Mutilations.

Removal of one or other of the upper central incisors was the most common dental mutilation seen in this area. Of the 42 mature males examined (all initiated), 27 had had this ceremonial extraction, 38 teeth having been lost in this way. Of the 45 mature females, four showed apparent mutilations, but two had Broca (iv) and two Broca (iii) attrition, so that loss of those teeth could have been due to abscess formation from exposure of the pulp resulting from excessive attrition—quite a common occurrence.

TABLE V.

Mean Number of Defective and Missing Teeth per Subject by Age Groups.

Group.	Number of Subjects.	Teeth Present.	Defective and Missing Teeth.		Mean Defective and Missing Teeth per Subject.	
			Including Mutilations.	Excluding Mutilations.	Including Mutilations.	Excluding Mutilations.
I	19	286	1	—	0·05	0·05
		192 deciduous	26	—	1·73	1·73
II	15	168 permanent	15	15	1·0	1·0
III	19	535	32	32	1·68	1·68
IV	54	1665	253	241	4·68	4·46
V	33	833	378	353	11·46	10·03
Total	34 deciduous	478	27	—	0·79	
	106 permanent	3225	678	641	6·40	6·05

In the males, 17 upper left central incisors had been extracted, compared with eight upper right central incisors, three upper right lateral incisors, three upper left lateral incisors, and one lower right central incisor. There was no case amongst the young males of multiple dental mutilation (Figure V), but of the older adults, three had had two teeth removed (usually the two upper incisors), one had had three teeth removed and two had had four teeth sacrificed (four upper incisors).

TABLE VI.

Mean Number of Defective and Missing Teeth per Subject by Age Groups and Sex (Excluding Mutilations).

Group.	Males.		Females.	
	Number of Subjects.	Mean Number of Defective and Missing Teeth.	Number of Subjects.	Mean Number of Defective and Missing Teeth.
I	7	—	12	0·08
II	6	1·83 deciduous	9	1·67 deciduous
		1·0 permanent		1·0 permanent
III	5	2·4	14	1·43
IV	21	3·86	33	4·82
V	19	7·74	12	17·17
Total ..	13 deciduous	0·85	21	0·76
	51 permanent	4·84	68	5·80

Gingival and Periodontal Conditions.

Gingival Crevice.

The depth of the gingival crevice in Group I was 0·5 mm.; in Group II, with many erupting teeth, the average crevice depth was 2 to 3 mm.; whilst in Groups III and IV the average crevice depth was 1 mm. In Group V the average was 2·5 mm., the extremes being 1 mm. and 5 mm.

TABLE VII.
Surfaces Affected by Caries in Deciduous Dentition.

Group.	Site.					Kind.		
	Occlusal.	Mesial.	Distal.	Labial.	Lingual.	Pits.	Fissures.	Smooth Surface.
I	1	—	—	—	—	1	5	—
II	14	5	6	3	7	6	10	17
Total ..	15	5	6	3	7	7	15	17

Gingivæ and Periodontal Condition.

In general, the condition of the gingivæ of the children in Groups I, II and III was excellent, presenting a firm, well-stippled appearance—pinky-red to dark brown according to the density of pigmentation. A small percentage of the younger children showed very slight chronic marginal gingivitis associated with food debris, while others presented slight hyperplastic gingivitis.

Group IV, apart from two individuals, presented healthy gingivæ, except in areas where some teeth had large deposits of calculus; in these, chronic marginal gingivitis was present, of remarkable mildness considering the quantity of calculus. Y40 and Y41 were the exceptions. Y40 presented generalized gingivitis with pocket formation, while Y41 exhibited advanced periodontal disease—the gingivæ bled easily, pockets of 4 mm. were present throughout the mouth, and movement could be initiated from all teeth, especially both lower second molars, which were very mobile.

In Group V all natives exhibited at least the chronic marginal gingivitis; but it was mild in most cases, although calcareous deposits and food debris were very marked in the majority. Eight cases of advanced periodontal disease with evident mobility of the teeth were noted; five of these subjects were females. Many had the majority of the roots exposed.

Calculus.

Calculus deposits did not become evident until adolescence—i.e., 12 to 18 years—at which stage small amounts of the soft supragingival type of calculus were laid down mainly on the buccal aspect of the upper molars. In Group III 63% of subjects showed calculus deposits, mainly very mild; 85% of Group IV showed calculus, mainly as mild to medium deposits. In Group V, 100% of subjects showed the presence of calculus, usually as either heavy or gross deposits.

Type of Calculus.—In general, the calculus deposited was of the soft supragingival variety, which in some cases, because of hyperplastic overgrowth, became subgingival although of the soft type. Hard subgingival calculus was noted in quite a large percentage of the older natives (Group V); but here difficulty was experienced in definitely establishing its presence, because of concidental deposits of soft calculus and large quantities of food debris. In general calculus was found on the lingual, interproximal, and labial aspects of the lower anterior teeth (Figure VI).

Table IX shows the incidence of calculus by age groups.

Pigmentation.

Of the 143 natives examined, only 32 did not show pigmentation, and of these 18 belonged to Group I.

Pigmentation was observed in only one case in Group I, being situated on the mid-line of the palate. In Group II mild pigmentation was noticed, mainly gingival (anteriorly, especially round the lower canines); but palatal and mucosal pigmentation was also observed in 11 of the 15 (Figure IV).

There was a slight increase in the average amount of pigment present in Group III, in which 14 of the 19 had pigment. The average grading for this group was: gingival > mucosal = palatal. Group IV presented increased mucosal depositions compared with Group III, the average being: gingival = mucosal > palatal. Group V showed a reduction in the average gingival pigment.

The most common sites for the pigment in these three areas were: (i) around the lower canines and upper central incisors; (ii) in the centre of the buccal pad; (iii) in the midline of the palate.

An arbitrary classification of intensity of pigmentation, based upon inspection of colour films, is illustrated diagrammatically in Figure II.

Hypoplasia.

In the young, hypoplasia was apparent in a quite large percentage of subjects (see Table X), occurring mainly in the first and second molars in the deciduous dentition, and mainly in the first molars in the permanent dentition. After this, the order of frequency was first premolars, canines, second molars and then central incisors.

This condition varied from simple defects in natural formation—e.g., a curved line on all the canines in Y86—to gross hypoplasia, most frequently seen in the first and second molars. Here the usual picture was one in which most of the buccal aspect was affected because of collapse of the matrix at its formative period. This could be due to any of the known causes—trauma, infection, heredity,

TABLE VIII.
Surfaces Affected by Caries in Permanent Dentition.

Group.	Site.					Kind.		
	Occlusal.	Mesial.	Distal.	Labial.	Lingual.	Pits.	Fissures.	Smooth Surface.
II	12	2	3	1	—	10	5	4
III	22	2	2	7	2	28	3	4
IV	86	55	53	57	7	122	9	117
V	19	51	62	8	6	16	10	113
Total ..	139	110	120	73	15	176	27	238
		230						

general diseases of genetic or idiopathic origin, or trophic disturbances.

Childhood illnesses in this area common to most aboriginal children include gastro-enteritis, chronic ear and eye infections and chronic nasal discharges. In addition, such common infectious diseases of Europeans as measles, scarlet fever, mumps, diphtheria, chicken-pox and tuberculosis also occur. Many of these are of significance as causative factors of hypoplasia.

TABLE IX.
Calculus.

Group.	Nil.	Slight.	Mild.	Medium.	Severe.
I	19	—	—	—	—
II	15	—	—	—	—
III	7	10	2	—	—
IV	8	25	15	4	2
V	—	8	4	12	9

Hypocalcification.

The incidence of hypocalcification by age groups and degrees of severity is shown in Table XI. The upper central incisors were the most frequently affected, the first permanent molars came next and then the upper lateral incisors. In Group II, seven of the eight showing hypocalcification, and in Group III six of the 13 similarly affected, exhibited the condition in all the teeth. It is of significance that in the deciduous dentition there was only one case in which hypocalcification—of a very mild grade—was noted, that being on two lower first deciduous molars. But in Groups II and III, the percentages of natives exhibiting the condition had increased to 53·3 and 76·3 respectively.

TABLE X.
Hypoplasia.

Group.	Number.	Nil.	Mild.	Severe.	Percentage Showing Hypoplasia.
I	19	13	4	2	31·6
II	15	6	7	2	60·0
III	19	12	5	3	42·1
IV	54	46	7	—	13·0
V	33	31	1	1	6·1

Variations.

Tooth Anatomy.

Tooth anatomy is discussed from the point of view of the incidence of peg-shaped incisors and extra cusps.

Two cases of peg-shaped incisors were noted.

The tubercle of Carabelli was observed in 15% of the natives. An additional mesio-buccal cusp was noted on four lower third molars. Accentuation of the lingula in lateral incisors to form another cusp was evident in two cases.

Three cases of very poorly developed third molars were also present.

Impacted, Unerupted and Displaced Teeth.

Eight cases of assumed (later confirmed radiologically) unerupted impacted third molars were observed. In addition, one first premolar and one second premolar were impacted. There were two cases of rotated lower second premolars, one case of lingual displacement of a lower permanent first molar and seven cases of over-eruption due to lack of opposing teeth.

Supernumerary Teeth.

One erupted supernumerary distal to (i.e., behind) the upper right third molar was noted. (See also radiological observations.)

Missing Teeth.

Two upper third molars and one lower third molar were missing, in two different subjects.

Erosion Cavities.

Erosion cavities were observed in the anterior teeth of one subject.

Space between Upper Central Incisors.

A space between the upper central incisors was observed in three cases, in two of which the space was due to the presence of large central frena. A large maxillary diastema, opposite the left lower canine, was also observed (Figure VI). This condition has been recorded before in aborigines (see Abbie and Adey, 1955).

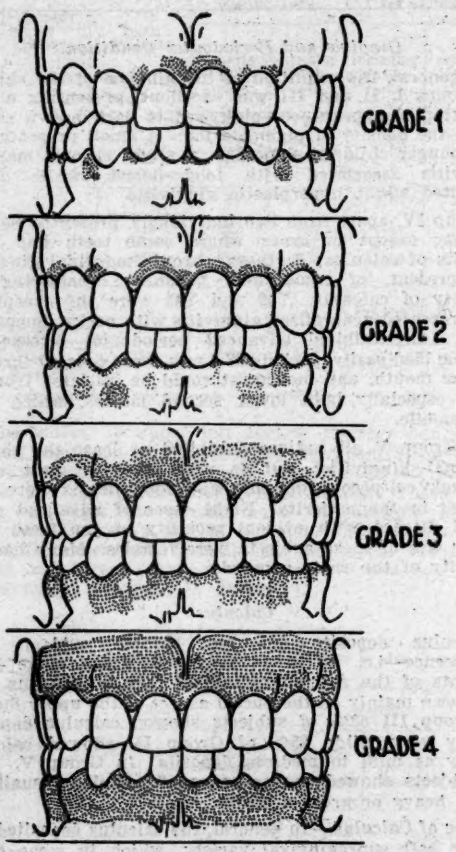

FIGURE II.
Diagrammatic illustration of the spread of gingival pigmentation in successive age groups.

Microstoma.

Microstoma was observed in Y63—a woman, aged 65 years, whose oral opening was very small in comparison with the size of her jaws and with the size of the opening in other aboriginal women.

Cleft Palate.

Y37 was a most interesting subject, for he presented a cleft in the soft palate on the left side—no uvula was present. His palate was very shallow. Also he showed gross calculus deposits on all surfaces of all teeth. His upper right central incisor was in traumatogenic occlusion —a rarity among these people, whose gross attrition

minimizes the chances of this condition. Many of these conditions will be discussed in greater detail in the second paper.

Radiological Observations.

The state of the dentition in respect to caries and restoration has been included under the section dealing with caries.

The ages at which the apices of the premolars and molars of the permanent dentition close are rather difficult to pinpoint, but the following represents the most accurate assessment possible from the numbers examined: first molar, closed by eight to nine years; first and second premolars, closed by 12 years; second molar, closed by 13 years; third molar, closed by 19 years.

TABLE XI.
Hypocalcification.

Group.	Number.	Nil.	Mild.	Medium.	Severe.	Percentage Showing Hypocalcification.
I	19	18	1	—	—	5·3
II	15	7	4	2	2	53·3
III	19	6	9	1	3	76·5
IV	54	48	6	—	—	11·1
V	33	31	2	—	—	6·1

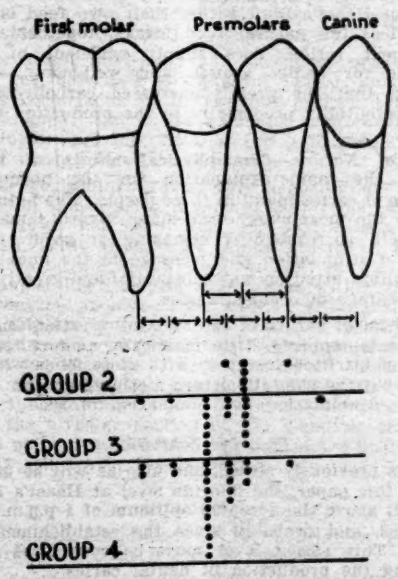

FIGURE III.
Diagram to show the position of the mental foramen relative to the apices of permanent teeth. Each dot represents an individual. With advancing age, the forward drift of the teeth produces an apparent backward drift of the foramen.

Position of Mental Foramen.

The position—or rather the changing position—of the mental foramen in relation to the apices of the teeth by age groups is diagrammatically represented in Figure III. In Group II the mean position is midway between the first and second premolars, whilst in Group III the foramen tends to be opposite the apex of the second premolar, the mean position being two-thirds of the distance between the premolars, and towards the second premolar. In Group IV the mean position is opposite the apex of the second premolar.

Extremes in level vary from a point midway between the canine and first premolar to one opposite the apex of the first molar.

Condition of Alveolar Bone.

Because of the extremely vigorous nature of the natives' mastication, and the consequent attrition, the investing alveolar bone was in all cases dense. In general, the investing bone presented the usual wire-netting appearance with the alveolar bone network closely interlaced. The alveolar crest was well marked, and a definite continuous lamina dura was present, with no signs of resorption.

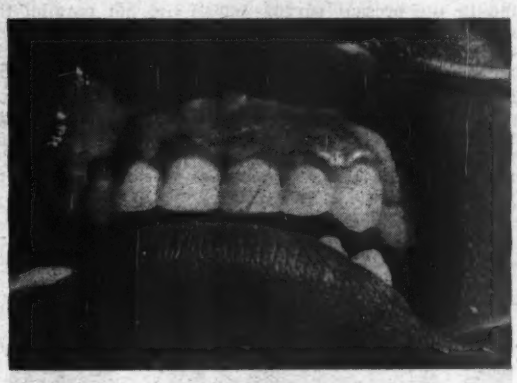

FIGURE IV.
Gingival pigmentation in a young adult male.

In subjects who had lost teeth, e.g., Y45, from whom a first molar had been extracted, resorption of alveolar bone was negligible, whilst the planes of the alveolar bone had become orientated parallel to the surface, i.e., at right angles to the direction of any applied force. There was a definite cortical layer on the surface of the alveolar bone in this region.

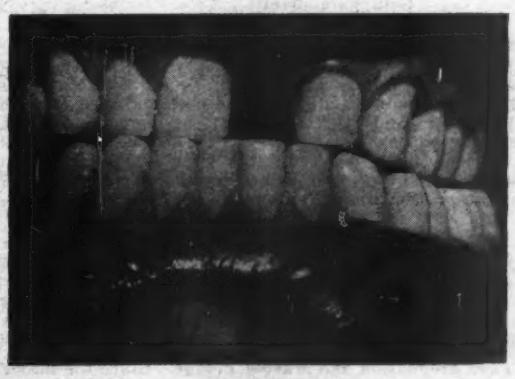

FIGURE V.
Ritual extraction of upper left central incisor in a young adult male.

Variations.

Only one periapical area was noted, that being around a lower first molar. From the X-ray films three impacted third molars were noted, two being mandibular, one maxillary. One mandibular was impacted vertically below the distal bulge of the second molar, the other on the mesioangular aspect. The maxillary impaction was vertical.

Two unerupted supernumeraries were revealed distal to upper third molars. There was one impacted upper first

premolar and one partially impacted lower second premolar, the roots of the preceding second deciduous molar still remaining unresorbed.

DISCUSSION.

Dental Caries.

The incidence of dental caries in all age groups has been found to be greatly increased on Campbell's (1925) original findings in nomadic natives of the pre-European era. Comparisons with work by Campbell (1934), Barrett (1953) and Cran (1955) reveals a diversity of results. All disclose a very low incidence of caries in Group I. The sudden increase in incidence at the mixed dentition stage found in the present survey (60%) did not parallel the findings of Campbell, Cran or Barrett, the last of whom found an increase of from 3·7% to only 30·5% in subject incidence in the transition from Group I to Group II.

The pattern of increase in tooth incidence in the adult corresponded with Barrett's results. He found that in the age range 19 to 29 years, 6·3% of the teeth showed caries, the mean being 2·0 carious teeth per subject rising to 13·9% in the age group to 49 years (mean carious teeth, 4·0), while the aged showed 14·0% with a mean of 3·4 carious teeth. These figures agree with the present results, since the over-all percentage for both estimations for complete adult dentitions has been recorded as 12·05% (this paper) and 11·4% (Barrett) for tooth incidence, and 3·2 (this paper) and 3·1 (Barrett) for the mean number of carious teeth per subject. Cran (1955) throughout found much lower D-M values for all groups than either Barrett or the present survey.

FIGURE VI.
Maxillary diastema opposite lower canine. All upper teeth were present. Note calculus.

Relative Effects of Predisposing Factors.

Certain factors are considered to predispose to dental caries in Europeans. By comparing the incidence of caries in natives exhibiting such recognized predisposing factors, one can more readily estimate their relative importance to either people.

Food Stagnation and Mucoid Plaque.—Since the majority of the mature natives exhibited a mucoid plaque on most teeth, and food stagnation interproximally and gingivally of considerable degree, the resultant predisposition to caries would by civilized standards be enormous. As the quantity of carbohydrate consumed by aborigines increases in succeeding years the natives may finally reach the civilized stage where such a mucoid plaque will be of considerable caries potential.

Irregularity of Teeth.—This is directly related to the foregoing, but in aborigines, because of interproximal attrition, the crowding of teeth quite frequently seen in the younger natives is usually eliminated by the time of completion of the permanent dentition. However, the early dental crowding is not associated with any increase in caries incidence. Hence, although crowding of the teeth increases the liability to food stagnation, it does not materially predispose to the occurrence of caries in the young aboriginal, who seems, under the present conditions, to possess a relative immunity until the onset of maturity.

Hardness of the Teeth.

No histological examination of the teeth was undertaken, but the appearance and the effect of the onset of caries make me agree with the findings of Cran (1955), who made the following statement:

> The histological structure of the aboriginal teeth is no better than, and if the accentuation of the perikymata is a reliable indication of histological defects, possibly not as good as, that of civilized subjects.

Thus tooth structure in no way gives the native an advantage over civilized Europeans; it predisposes him to gross caries if the exciting factors are present.

Composition of Saliva.

The consistency of the saliva in general was thick and ropy, thus tending to cause the accumulation of food on the surface of the teeth.

Physical and Chemical Nature of Foods.

The extent to which all these factors manifest themselves is, of course, dependent on the physical and chemical nature of the foods. This has been discussed in detail by Campbell (1928, 1938, 1939).

Chemical Nature.—Although the extent to which the natives at Haast's Bluff gather their own food is limited, nevertheless the natural food intake, together with the Government ration, does supply sufficient of all the essentials for bodily growth and well-being, with the exception that the greatly increased carbohydrate component potentially predisposes to the production of dental caries.

Physical Nature.—The physical nature of the food supplies the main explanation for the normally low incidence of caries found in these people. The natural food, and also the meat supplied, demand vigorous masticatory effort with corresponding cleansing or scouring of the teeth, and stimulation and massage to the gum margins. The resultant attrition was substantial and greatly reduced the percentage of occlusal caries.

The physical nature of the remaining ration and bought supplements required little masticatory effort. A certain amount of attrition resulted (with those foods which were cooked), but the over-all picture disclosed greater tendency towards accumulation and stagnation of food.

Fluorine Inhibition.

As was previously stated, and also as will be mentioned later in this paper, the fluoride level at Haast's Bluff has been well above the accepted optimum of 1 p.p.m. since at least 1953, and probably since the establishment of the station. This raised level must be considered a factor inhibiting the production of dental caries.

Hypocalcification.

Disturbances in mineralization, in the absence of any other obvious ætiological factors—e.g., low calcium and low phosphorus intake—may be assumed to be due to the effect of fluorine in the water intake. This is thought to act in either of two ways to produce disorder. De-Eds (1941) has suggested that there may be a deposition of calcium fluoride in the matrix instead of a calcium phosphatase disturbance. Irving (1943) has suggested that fluoride acts primarily by altering the calcium-phosphorus composition of the blood, and that the changes in mineralization are variants of the normal process.

The fluorine content of bore water at Haast's Bluff has, according to all available records, varied between 1·8 p.p.m. (present value) and 2·2 p.p.m. This record extends back to August, 1953. However, a value of 2·2 p.p.m. was determined by the Department of Animal Industry of the

Northern Territory Administration at some date prior to January, 1954. It is difficult to establish any definite correlation between the large percentage of natives showing hypocalcification in teeth and the amount of fluoride in the water, as dates of known estimations prior to August, 1953, are unprocurable. This is due to the fact that only in recent years have fluoride estimations become a routine procedure in all water sampling. Mr. H. N. J. Hodgson, of the Engineering and Water Supply Department, has stated that a great number and variety of water analyses for fluoride during the past 14 years in South Australia have shown that the variation from any mean has been no greater than 20% (personal communication).

On this basis, one could assume that the fluoride value at Haast's Bluff had not varied from the mean by a value of even 25%—i.e., by approximately 0·5 p.p.m.—for at least the last ten years. As the mean of known estimations was 2·3 p.p.m., one may reasonably presume that the fluoride value has always been well above 1 p.p.m., and that the observed hypocalcific condition is probably related to this raised level.

The high incidence of gastro-enteritis has been cited by Cran (1955) as increasing the amount of fluoride concentrate in the stomach and so raising the incidence of hypocalcification.

Position of Mental Foramen.

The apparent backward move of the mental foramen is, of course, purely a relative observation, and in assessing it one must consider the factors governing this relative position. These are: (i) attrition and mesial (forward) migration of teeth, (ii) normal horizontal growth and (iii) buckling of the main mandibular core.

Interproximal attrition, and consequent mesial migration, as first described by Campbell (1925), must be considered a major factor in this apparent backward movement of the foramen; that is, the changing relationship is due largely to forward migration of the teeth rather than to posterior movement of the foramen.

Normal horizontal growth of the mandible must be looked upon as occurring in two parts, alveolar and basal. Murphy (1957) has shown that the Australian aboriginal differs materially from other ethnic groups in the tooth relationship of the mental foramen and in width of the mental angle. He has also shown that within the group a high degree of association exists between these two features, and has stated that "both are explicable in terms of the Australian alveolar arch being more forwardly placed on the basal arch of the mandible". Since we can consider the mental foramen as belonging to the basal part of the mandible, this differential forward growth between the alveolar portion of the mandible relative to the basal portion results in the placing of the teeth further anteriorly relative to the foramen.

Summary.

1. The dental condition of 145 aborigines from the neighbourhood of Haast's Bluff is described.

2. The incidence of dental caries and the D–M values by age and sex are discussed in detail.

3. Comparisons are made with previous data. The incidence of caries, although much lower than in civilized Europeans, was much increased over that in nomadic natives. While aborigines seem to preserve some immunity to caries during adolescence, once caries sets in it progresses rapidly.

4. Gingival condition and pigmentation are described and defined. In general, the gingival condition was very good and there were relatively few cases of periodontal disease.

5. Dental mutilations in this area are outlined, and there is also a list of variations.

6. The radiological findings in 64 lateral X-ray films have been examined, and from them the changing relative position of the mental foramen is traced, as well as the ages of closure of the apices of premolar and molar teeth. Radiological variations are also listed.

7. The incidence of hypoplasia and hypocalcification is detailed along with possible causes.

8. These findings are discussed in relation to environment, including diet.

Acknowledgements.

I am indebted to Professor A. A. Abbie for making this material available for study, for constant help and advice during preparation of the paper and for seeing it through the press. Professor T. D. Campbell, Mr. M. J. Barrett and Mr. J. A. Cran afforded useful discussion and advice. Miss G. D. Walsh prepared the illustrations. Thanks are due also to the Honourable Paul Hasluck, Minister for Territories, to His Honour the Administrator of the Northern Territory, and to the Director of Welfare, for affording access to the Haast's Bluff Settlement; to the Commonwealth and South Australian Railways Departments for providing free transport for heavy equipment; and to the various members of the Haast's Bluff Settlement for ready assistance.

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POISONING BY CARBROMAL AND BROMVALETONE.

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EACH year the Victorian Medico-Legal Laboratories deal with two or three fatal cases of poisoning by the bromureides, carbromal and bromvaletone, derived from proprietary tablets which are obtainable in Victoria without medical prescription. Seven cases are listed in Table I, and two of these are described in detail.

Some of the problems associated with the toxicology of these bromureides are discussed.

CASE I.—The patient was a female, aged 54 years. At the inquest into her death, it was found that she died from an overdose of proprietary tablets containing carbromal, 3 grains, and bromvaletone, 2 grains. Evidence was given that the deceased had been taking tablets of this particular brand for about five years, and at the

time of her death there were numerous empty and partly-used packets hidden about her home. Although her husband had made attempts to withhold these tablets, she managed to obtain further supplies by ruses suggestive of a true addict. Witnesses stated that under the influence of these tablets she was quite unable to stand or walk properly. The fatal dose was probably 46 tablets taken during the day and evening prior to her death. Death occurred before 7 a.m. when she was discovered in her bed.

Routine post-mortem examination did not reveal the cause of death. Mild œdema of the lungs, insufficient by itself to account for death, was consistent with death from an overdose of the bromureides found on analysis of the organs. The concentration of bromine found in the liver was 502 parts per million (mg./kg.).

CASE II.—The patient was a male, aged 45 years. The deceased was initially found at about 9.45 p.m. in his bath, and appeared to be in a drunken stupor. Found near him was a bottle containing whisky, in which was suspended the equivalent of 72 powdered bromvaletone tablets. He was removed to his bed by a friend, who assumed he was drunk and therefore did not summon medical aid. At 8.30 a.m. on the following morning, he was found in a natural sleeping position with a small amount of vomit on his pillow. An examination showed that he was dead. Inquiries revealed a history of alcoholism, and that he had previously attempted suicide by slashing his wrists with a razor blade.

No obvious cause of death was found on post-mortem examination. The coronary vessels were mildly atheromatous, and there was mild œdema of the lungs. These findings were insufficient to account for death from natural causes. Over 35 grains of bromvaletone were found in the stomach of the deceased, and the serum bromine content was 118 mg. per 100 ml. present as organic bromine compound. No inorganic bromide was detected in his serum.

In addition to fatal cases of poisoning by carbromal-bromvaletone mixtures, it is usual for our laboratories, at the request of hospital authorities, to analyse blood and urine samples in several non-fatal cases each year. During the three years 1954 to 1956, three of the drivers arrested by the Victorian police for driving under the influence of alcohol were found to be sedated with proprietary tablets containing bromvaletone and carbromal.

The first case described above is more or less typical of the cases of bromureide poisoning, in which there is usually a history of a long period of self-medication with these or other hypnotics. The layman believes that the proprietary mixtures under discussion are harmless, and is confirmed in this opinion by the fact that he can obtain unlimited quantities without a prescription.

Queensland is the only Australian State in which a prescription is necessary to obtain these drugs, and this is cancelled unless a repeat is ordered. The unrestricted sale in Victoria of mixtures containing carbromal and bromvaletone has created problems for medical practitioners.

References in the literature to the toxicity of carbromal and bromvaletone are comparatively few, and toxicologists are still faced with some difficult questions. For example, what are the lethal doses of these drugs, and of what value are the findings of the analyst?

The answers to these questions are also important to the pathologist, who does not find anything characteristic at post-mortem examination when death has occurred from an overdosage of these drugs.

Carbromal.

One of the most interesting articles on the toxic effects of carbromal was published by Magnussen (1947), and deals with cases of acute poisoning, in which the patients were admitted to the psychiatric division of the Copenhagen Municipal Hospital. This author points out that, as the toxic dose of carbromal is only seven times its maximum therapeutic dose, it can be quite as dangerous as stronger hypnotics. It was the third most common toxic agent he encountered in his work during the years 1945 and 1946. Martindale (1958) has reproduced an abstract of Magnussen's paper, and draws attention to two deaths that he recorded. In one case 10 grammes (about 150 grains) proved fatal, and in the other, death occurred after the ingestion of 25 grammes (about 380 grains). Ackroyd (1948) found an idiosyncracy to carbromal by two subjects in whom "Sedormid" (allyl isopropyl acetyl urea) had previously produced thrombocytopenic purpura. One of these patients also developed purpura with bromvaletone. In personal communications three medical practitioners have told me of cases of purpura occurring in patients taking carbromal.

Experiments on animals are reported in an article by Flury and Zernick (1935), who record a M.L.D. (oral) of 350 mg./kg. for cats. By comparison, Krop and Gold (1946) found that the LD_{50} of phenobarbitone given orally to cats was about 175 mg./kg.

Another role of carbromal in fatal cases of poisoning is illustrated by the data in Table II, where it is seen that the mean concentration of pentobarbitone found in liver tissue from persons who died from overdosage is

TABLE I.

Fatal Cases of Poisoning with Proprietary Bromureides (Victoria, 1956 to 1958).

Case Number.	Sex and Age. (Years.)	Bromureide Taken.	Amount Believed Ingested. (Grains.)	Time of Ingestion to Time of Death. (Hours.)	Treatment.	Amount of Bromureide Found in Stomach. (Grains.)	Bromine Concentration in Liver. (Mg./kg. or Parts per Million.)	Straight-Chain Ureide Concentration in Liver. (Mg./kg. or Parts per Million.)	Post-Mortem Findings of Note.
52/56	M. 33	Carbromal and bromvaletone.[1]	360 240	Less than 12	Hospital, stomach wash.	1·4	Not estimated.	140	Brain congestion.
724/56	F. 39	Carbromal and bromvaletone.[1]	Not known.	Not known.	Nil	Nil	340	6	Marked œdema of lungs.
236/57	F. 61	Carbromal and bromvaletone.[1]	Not known.	12	Hospital, stomach wash.	Nil	140	26	Brain congestion, bronchopneumonia, œdema of lungs.
364/57	F. 57	Carbromal and bromvaletone.[1]	Not known.	12	Hospital, stomach wash.	Nil	470	17	Brain congestion, marked œdema of lungs (slight coronary atheroma).
549/57[2]	F. 54	Carbromal and bromvaletone.[1]	138 92	12 to 24	Nil.	0·2	502	22	Slight œdema of lungs.
184/57	M. 36	Carbromal and bromvaletone.	540 360	Less than 12	Nil.	600	80	10	Lung œdema (slight coronary atheroma).
412/58[3]	M. 45	Bromvaletone.	140	Less than 12	Nil.	35	1195	32	Mild œdema of lungs (slight coronary atheroma).

[1] Same brand of proprietary tablet. [2] Case I in text. [3] Case II in text.

42·3 ± 18·3 parts per million (i.e., mg./kg.), whilst the concentration of pentobarbitone found in fatal cases of poisoning by mixtures of pentobarbitone and carbromal is 26·7 ± 9·9 parts per million. The difference in these figures is statistically significant.

Bromvaletone.

Bromvaletone is generally regarded as a harmless hypnotic and is described as such in "The Physician's Index of Australia and New Zealand" (1956). Martindale (1958) does not list any toxic effects of this drug. One of the earliest investigators of bromvaletone was Sollmann (1908), who carried out experiments on cats, and compared the toxicities of chloral hydrate, "Isopral" (trichloroisopropyl alcohol) and bromvaletone ("Bromural"). This last hypnotic had only recently been placed on the local American market. Sollmann found a minimum lethal oral dose of 450 mg./kg., and concluded that in a dose exceeding the normal therapeutic amount, bromvaletone could be just as dangerous as chloral hydrate. He stated that bromvaletone could not be called absolutely harmless.

TABLE II.

Fatal Cases of Poisoning by Pentobarbitone and Pentobarbitone with Carbromal.
(Victorian Medico-Legal Laboratory, 1956 to 1958.)

Hypnotic.	Number of Cases.	Concentration of Pentobarbitone in Liver. (Mg./kg.)		
		Minimum.	Maximum.	Mean.
Pentobarbitone only	16	19	88	42·3±18·3
Pentobarbitone with carbromal ..	11	17	47	26·7± 9·9

In cases of poisoning by barbiturates, the statistical treatment of the results of previous analyses, such as that carried out by Copeman (1954), is of value to the toxicologist, especially when the cause of death is not apparent on routine post-mortem examination. Such an approach has not been possible in cases of poisoning by the bromureides, carbromal and bromvaletone. Over the years chemical analysis of post-mortem material for the bromureides has depended on the detection of bromine in organs such as the liver, and Bamford (1951) suggests that such tests may be the only evidence of poisoning by these drugs.

In these cases the detection of bromine alone does not allow any conclusion to be reached as to the nature of the original compound, except where carbromal or bromvaletone is identified in the gastro-intestinal tract. This bromine could have originated from potassium bromide. Our only other finding in cases of poisoning by carbromal or bromvaletone is the isolation from the body tissues, by the Stas-Otto process,[1] of a straight-chain ureide which, unlike the parent compound, has no bromine substitution. We have encountered these straight-chain ureides only in cases in which bromureides had been taken by the deceased. The author has found that the ureide isolated from persons poisoned by carbromal is quite distinct from that isolated from persons poisoned by bromvaletone and also by "Sedormid".

In each case of bromureide poisoning encountered in these laboratories, the concentrations of bromine found in the liver, kidney and serum have been low compared with the figure of 200 mg. of sodium bromide per 100 ml. of serum quoted by Harrison (1949), which he claims is the concentration that could indicate inorganic bromide intoxication. The clinical biochemist should note that in the estimation of bromide in blood and tissues in cases of bromureide poisoning, it is necessary to use special ashing techniques.

[1] This method of isolation of organic drugs from tissue is described by most medico-legal text-books, including Bamford (1951), page 151.

For the toxicologist it would appear that the identification and estimation of a metabolite may be the best method of determining whether a person has been poisoned by carbromal or by bromvaletone. Investigations along these lines are being carried out at these laboratories.

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THE USE OF RADIOACTIVE IODINE (I^{131}) IN THE DIAGNOSIS OF THYROID DISORDERS.

By BRYAN HUDSON AND F. I. R. MARTIN,
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ALTHOUGH used initially for the treatment of patients with thyrotoxicosis, radioactive iodine (I^{131}) has since been applied extensively to the diagnosis of thyroid disorders. Many different parameters of thyroid function have been measured with I^{131}, but some controversy still exists as to the most efficient means by which this may be assessed.

The use of I^{131} in diagnosis depends upon the ability of the thyroid to concentrate inorganic iodide from plasma and convert it to thyroid hormone. Accordingly, tests of thyroid function with I^{131} are designed to measure the capacity of the thyroid either to concentrate iodine or to convert the iodine isotope into circulating hormone.

Some of the tests that employ I^{131} in diagnosis require complicated apparatus and skilled technicians, and involve the patient in many measurements. In clinical practice it is essential that any method should, while preserving accuracy, possess the advantage of being relatively simple, inexpensive and rapid. In general, however, no single test gives a completely correct classification of all patients with normal or abnormal thyroid function, so that a combination of two or more measurements is desirable (Werner *et alii*, 1950; McConahey *et alii*, 1956; Bruck and Goldberg, 1958).

The purpose of this paper is to describe our experience with the use of I^{131} in diagnosis, using as indices the uptake of radioactive iodine by the thyroid gland and the concentration of radioactive protein-bound iodine (PBI^{131}) in plasma after administration of a tracer dose.

Materials and Methods.

Patients.

Three hundred and twenty-one persons, including 50 normal controls, have been investigated. The patients, referred because of suspected thyroid disease, have been examined by one or other of us. Their classification into hypothyroid, euthyroid or hyperthyroid categories has been based on clinical examination of all patients, and, when diagnosis was in doubt, determination of basal metabolic rate and protein-bound iodine. In some cases these investigations have been repeated after weeks or months of observation; in others, final evaluation has been possible only by the use of thyroid-stimulating hormone or triiodothyronine.

Radioactive Iodine.

All tracer doses have consisted of 50 μc. of I^{131}. This has been supplied by the Commonwealth X-ray and Radium Laboratories as carrier-free radioactive sodium iodide in 2·5 ml. of weak sodium thiosulphate solution.

Administration of Dose.

Every dose has been administered orally between one and two hours postprandially, the patient having been instructed to take a light meal prior to the test.

Detecting and Counting Apparatus.

Surface Counting.—This was performed with a thallium-activated sodium iodide crystal. The crystal was shielded by a lead cone, similar to that recommended by Oddie *et alii* (1955). The arrangement of the lead shield, crystal and photomultiplier tube is shown diagrammatically in Figure I. Preliminary experiments had shown that

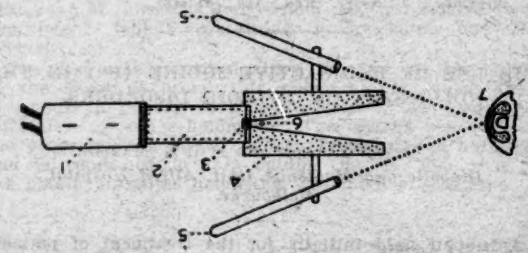

Figure I.
Diagrammatic representation of counting apparatus.

when assembled in this fashion and placed 35 cm. from the neck of the patient, the crystal "saw" a circle of neck, approximately 10 cm. in diameter.

Precision in directing the crystal to the centre of the thyroid (determined by palpation) and in measuring the correct distance (35 cm.) from the crystal to the surface of the neck was obtained by an optical system built into the machine. Fine crossed wires on the front aperture of the lead shield, illuminated by a light source in front of the crystal, permitted accurate centring; placed laterally on the lead shield was a pair of lamps so arranged that their beams of light converged and formed a single point at the required distance. When the apparatus was in operation, the light source in front of the crystal was removed.

This apparatus was supported by the upright arm of a converted X-ray machine, and was able to be moved freely in both vertical and horizontal directions. This machine had been converted further for the purpose by the attachment of a dental chair with neck rest, so that the whole became a mobile unit. The complete assembly, shown in Figure II, allowed measurements to be made with the patient sitting with the neck firmly supported by the neck rest.

Calculation of Uptake.—The amount of radioactivity in the neck at any given time was compared with that of a standard reference source of I^{131} of known activity. This reference was contained in a paraffin wax "phantom neck". Preliminary experiments had shown that the amount of radioactivity in extrathyroid tissues was approximately that measured in the patient's thigh, so that in each instance, due allowance was made for this extrathyroid radioactivity.

Plasma Counting.—Plasma from heparinized blood, collected at 48 hours after the administration of the tracer dose, was prepared by a method similar to that described by Clarke and Aujard (1954), except that the estimation was performed with 1 ml. of plasma.

The activity of the dried plasma was measured by counting each sample in duplicate for 30 minutes with an organic quenched, thin-window G-M tube, with the sample in a lead castle (Nuclear, Chicago). This was compared with that of a known amount of I^{131} prepared as silver iodide on a copper reference disk. The amount of I^{131} present was expressed as a percentage of the administered dose per litre of plasma.

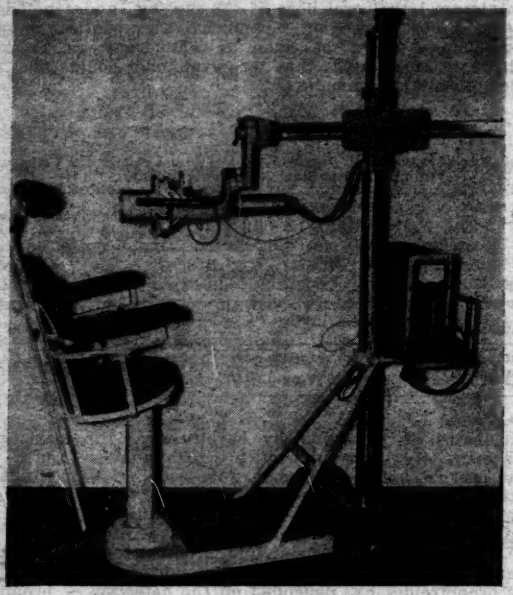

Figure II.
Neck-counting apparatus in use.

Counting Apparatus.—A binary scaler (Model 182, Nuclear, Chicago) was used for recording surface and plasma counts.

Results.

Uptake of Radioactive Iodine.

The uptake of radioactive iodine was determined in 172 cases[1], in 75 at four and 24 hours, in 25 at 24 hours only and in 72 at four hours only after the administration of the dose. The results in relation to the status of the patient are depicted in Figures III and IV.

Twenty-four Hour Uptake.—The mean and range of values for euthyroid, hyperthyroid and hypothyroid patients are shown in Table I. Although the mean values differ significantly, Figure III shows that there is a considerable overlap between the groups. It is difficult to draw a sharp distinction between euthyroid and hyperthyroid patients. Placing this value at 60% classifies 12 (31·7%) of hyperthyroid patients as euthyroid, and 11 (18·9%) euthyroid patients as hyperthyroid.

Four Hour Uptake.—The mean and range of values for this parameter are shown in Table II. In hyperthyroidism these values are higher, and for euthyroid and hypothyroid subjects lower, than those obtained from 24 hour uptake. Figure IV shows that the overlap of uptake values is less

[1] In the remaining 149 cases the uptake was measured at 48 hours. As these results were found to be of little diagnostic value, they are not included.

at four hours than at 24 hours. If a value of 50% is regarded as the upper limit of normal, 14·8% of hyperthyroid patients are classed as euthyroid and 16·7% of euthyroid patients as hyperthyroid.

Comment.—Both at four and 24 hours, patients with hypothyroidism show low uptake values. If values below 10% are chosen to indicate hypothyroidism, two patients in each group fall outside this range. Three of these were patients with hypopituitarism, in whom some autonomy of

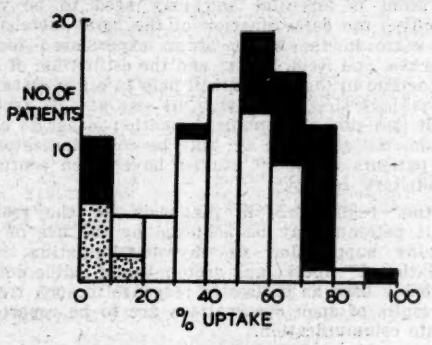

FIGURE III.

The distribution of results of 24 hour I^{131} uptake: solid columns, hyperthyroid; open columns, euthyroid; dotted columns, hypothyroid.

thyroid function frequently persists. The finding of very low uptake values (less than 10%) in patients with thyrotoxicosis is difficult to explain, but has been reported elsewhere (Chapman and Maloof, 1955). Three of these patients had a history of medication with iodine within three months prior to the test.

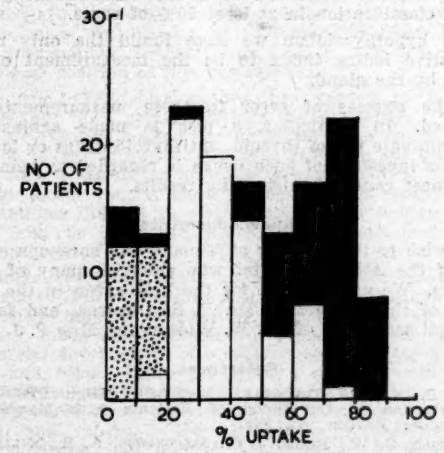

FIGURE IV.

The distribution of results of four hour I^{131} uptake: solid columns, hyperthyroid; open columns, euthyroid; dotted columns, hypothyroid.

Forty-eight Hour PBI^{131}.—This was determined in all 321 cases, and the results are shown in Figure V. If those patients who had been subjected to subtotal thyroidectomy or treatment with radioactive iodine are excluded from consideration, this procedure results in the misclassification of 10 (9·9%) of hyperthyroid patients, eight of whom also had uptake values of less than 10% at four or 24 hours, and 2·5% of patients with normal function. Abnormally high plasma PBI^{131} values were observed in 25 patients who were not thyrotoxic, of whom eight had previously been subjected to partial thyroidectomy and 12 treated with radioactive iodine.

In 16 hypothyroid patients, the plasma PBI^{131} value was in the euthyroid range, so that this procedure was not able to separate these two groups of patients.

TABLE I.

The Mean and Range of Values of 24 Hour Uptake of I^{131} (Percentage of Administered Dose).

Status of Subject.	Mean.	Range.
Euthyroid	47·9	18·0–83·8
Hyperthyroid	66·2	1·6–96·0
Hypothyroid	6·9	2·1–14·1

Discussion.

The measurement of I^{131} uptake by the thyroid at four or 24 hours, and of the plasma concentration of PBI^{131} at 48 hours after a tracer dose, are conventional procedures. Both are simple and fairly rapid, and the inconvenience to the patient is minimal. Admission to hospital is not necessary, and the patient is required on only three occasions, each for about 10 minutes. The limitation of these procedures is that either may incorrectly assess thyroid function. When they are applied in combination, however, the probability of misclassification is small.

TABLE II.

The Mean and Range of Values of Four Hour Uptake of I^{131} (Percentage of Administered Dose).

Status of Patient.	Mean.	Range.
Euthyroid	34·9	11·4–74·2
Hyperthyroid	69·4	1·5–88·5
Hypothyroid	5·8	1·3–18·5

Although measurement of the uptake of I^{131} at 24 hours has been held to give reliable separation of patients (Werner *et alii*, 1950), other reports suggest that an early uptake measurement is more satisfactory (Goodwin *et alii*, 1951; McConahey *et alii*, 1956; Clarke, 1958). In this series of patients, measurement of I^{131} uptake at four hours shows less overlap than at 24 hours. At both times, however, abnormally high values were observed in patients who were not hyperthyroid. In some, the prior administration of antithyroid drugs may possibly explain these findings (Foote *et alii*, 1952); but in others of this "high uptake" group no explanation is obvious. They can, however, be differentiated from hyperthyroid patients by the finding of a normal PBI^{131} value and by the use of triiodothyronine to suppress uptake.

Abnormally low values of four or 24 hour I^{131} uptake were observed in patients who, by other criteria, were thyrotoxic. Although there is no clear explanation for these findings, in some of the patients hyperthyroidism was associated with a large nodular goitre, and others had been given iodine within 12 weeks of the uptake measurement. In some of the former group, confirmation of the hyperthyroid state was obtained from an elevation of the plasma PBI^{131} value, but in the latter group this value was in the normal range.

We believe that it is important to stress the diagnostic difficulties that may be created by the use of iodine, antithyroid drugs or thyroid extract in patients with suspected thyroid disease. Confusion with respect to the interpretation of results of tracer studies with I^{131} has arisen most commonly in patients to whom one or the other of these drugs has been administered. Because of this and of the 92% to 95% reliability of properly controlled I^{131} studies,

we believe that the practice of subjecting patients to so-called therapeutic tests with these drugs is to be condemned. Attention is also drawn to the use of preparations not suspected to contain iodine; for instance, considerable delay and confusion were caused in two cases of thyrotoxicosis in which iodine-containing intestinal antiseptics had been given because of persistent diarrhœa.

In hypothyroid patients there is no real difference in the uptake measurements at four or 24 hours. The uptake at both times is low. In all patients it was less than 20% and in most less than 10%. This procedure is, therefore, reliable in the assessment of reduced thyroid function. Attention should be drawn to falsely low uptake values that occur in normal patients taking thyroid and the occasional use of this measurement in the diagnosis of thyrotoxicosis factitia (Farran, 1957).

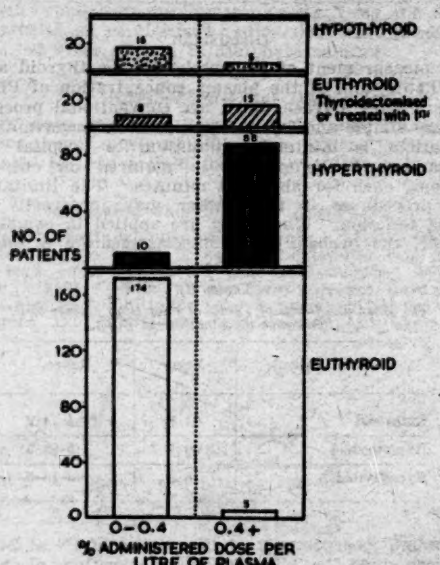

FIGURE V.

The 48-hour plasma PBI¹³¹ value as an index of thyroid function.

The estimation of plasma PBI¹³¹ concentration at 48 hours has been the most useful single procedure in the differentiation of hyperthyroid and euthyroid patients in this series. The value (0·4% of the administered dose per litre of plasma) at which the division between normal and increased thyroid function is made gives the most accurate classification and is similar to that described by others (Clarke *et alii*, 1955). On this basis, only 2·5% of euthyroid and 9·9% of hyperthyroid persons have been misclassified. Goodwin *et alii* (1951) and Silver *et alii* (1952) placed similar reliance on this parameter, the former claiming that it was the most useful single procedure in the diagnosis of hyperthyroidism, despite the need for the relatively high tracer dose (in excess of 30 μc.). However, the plasma PBI¹³¹ estimation is of no value in distinguishing between normal and reduced thyroid function, very similar values being obtained in both groups of patients. Furthermore, in both euthyroid and hypothyroid patients who have been subject to subtotal thyroidectomy, misleading results may occur, the plasma PBI¹³¹ values being frequently in the hyperthyroid range. This has also been observed in several patients in this series who, although euthyroid, had been treated with I¹³¹ for thyrotoxicosis. The observation and significance of high PBI¹³¹ values in such patients have been discussed previously (Blomfield *et alii*, 1951; Blom and Terpstra, 1953; Nodine *et alii*, 1955); and we would agree with Silver *et alii* (1955) that the I¹³¹ tests are not reliable after treatment of thyrotoxicosis by radio-iodine or thyroidectomy.

The purpose of this paper has been to describe our experiences with the use of I¹³¹ in the diagnosis of thyroid disorders. While we are convinced that this isotope serves a most useful function in this regard, permitting a correct classification of between 90% and 95% of patients, we would not discount other well-established methods by which a correct diagnosis may be reached. Careful clinical observation is essential and may need to be repeated frequently; the determination of the basal metabolic rate is of value in the hands of an experienced technician (Robertson and Reid, 1952); and the estimation of protein-bound iodine in the plasma will help in other cases. It has been our practice to restrict the use of this technically difficult and time-consuming estimation either to children, to whom the giving of I¹³¹ may be contraindicated, or to those patients whose I¹³¹ studies have given confusing or contradictory results.

Further refinements in diagnosis for the residue of difficult patients may be achieved by the use of triiodothyronine suppression or thyroid-stimulating hormone stimulation, I¹³¹ uptake and protein-bound iodine determinations being used as indices of response to such treatment. The results of such observations are to be reported in a separate communication.

Summary and Conclusions.

1. The use of radioactive iodine for the diagnosis of thyroid disease in 321 patients at the Alfred Hospital, Melbourne, is discussed; and the techniques used in estimating the uptake of iodine by the thyroid and the determination of plasma protein-bound radioactive iodine are described.

2. In suspected hyperthyroidism, the uptake by the thyroid gland four hours after an oral tracer dose of radio-iodine, and the level of plasma protein-bound radioactive iodine 48 hours after the dose, are used. These give a correct classification in at least 90% of cases.

3. In hypothyroidism we have found the only useful radioactive iodine index to be the measurement of the uptake by the gland.

4. The sources of error in these measurements are discussed. In particular, a plea is made against the indiscriminate use of thyroid, antithyroid drugs or iodides. Previous ingestion of such drugs is regarded as being the commonest cause of misleading results.

Acknowledgements.

We wish to thank other physicians and surgeons on the staff of the Alfred Hospital who referred many of these patients. We are grateful for the help given in the early stages of this project by Dr. P. R. Davoren, and for the technical assistance of Mr. W. Hudson and Mrs. P. J. Keen.

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THE USE OF A MICROHÆMATOCRIT CENTRIFUGE: A PRELIMINARY REPORT FROM AN OBSTETRIC AND GYNÆCOLOGICAL UNIT.

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THE estimation of the packed cell volume is of considerable importance in hæmatological investigations. There is a small degree of experimental error when precautions are taken to ensure accurate mixing and consistent aeration (Biggs and MacMillan, 1948), and it is this reproductibility of results, together with its use in the estimation of other values such as mean corpuscular volume, mean corpuscular hæmoglobin and mean corpuscular hæmoglobin concentration, which has led to the established position of this measurement.

The usual Wintrobe method has proved to be very reliable, but does present certain disadvantages. Corrections for trapped plasma are necessary even when centrifugation is carried out for one hour (Chaplin and Mollison, 1953), and to ensure an accurate measurement, it is necessary to obtain venous blood without undue stasis. This is sometimes difficult in obese women with inconspicuous veins, and is rarely attempted in infants and small children.

Methods requiring small amounts of blood have been used since 1915 (Epstein), but have not become more universally applied because of difficulties in obtaining capillary tubes of consistent bore, and also the lack of commercial development of a centrifuge to take such tubes. Guest and Siler (1934) developed a machine which has been modified, and reports of the use of a modified machine have appeared from McInroy (1954), McGovern *et alii* (1955) and others. Recently such a machine with capillary tubes has become available in England and Australia. This paper reports some preliminary investigations with a machine available in Sydney to assess its accuracy with both capillary and venous blood in comparison with the routine Wintrobe method.

Materials.

Tubes.

Capillary tubes, 75 mm. long and having an internal diameter of approximately 1 mm., are used. These are supplied by Hawksley and Sons, Ltd., London. Some of the tubes contain dried heparin for use for the collection of capillary blood, while others are plain, and are used when the blood already contains an anticoagulant. After use, the tubes are discarded.

The "Hawksley Microhæmatocrit Centrifuge".

The centrifuge (Figure I) consists of a head with 24 radial slots cut into it for the capillary tubes, the sealed ends resting against the rim at the periphery of the disc. The head has a closely-fitting, detachable cover to keep the tubes from being thrown out during centrifugation.

FIGURE I.
Microhæmatocrit centrifuge.

The centrifuge is driven by a high-speed motor, which develops 12,000 G and reduces the centrifuging time for complete packing to a maximum of five minutes. A preset, automatically-timed device controls the duration of centrifugation. Balancing is unnecessary.

The "Hawksley Microhæmatocrit Reader".

This reader (Figure II) will allow the accurate measurement of the packed cell volume with total fluid heights between 40 and 70 mm. The capillary tube is placed in

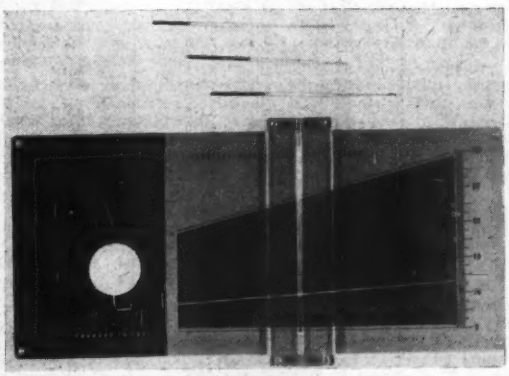

FIGURE II.
Microhæmatocrit reader.

the slot so that the base line of the reader intersects the base line of the red cells. The tube holder is adjusted so that the top line intersects the top of the plasma, and the middle line is moved to the top of the red cells. The packed red-cell volume is read on the scale.

Method.

Patients requiring routine investigations at the obstetric and gynæcological clinics were the subjects of study in this investigation.

Blood was obtained by finger-prick for capillary hæmatocrit estimation. When a free flow of blood was obtained, the heparinized tubes were filled by gravity to within

one or two centimetres of the end. Venipuncture was carried out without stasis for the venous hæmatocrit, and the blood was placed in heparinized containers.

The venous sample was well mixed and aerated, and the blood was placed in Wintrobe tubes and run into the plain capillary tubes.

The capillary tubes were sealed by having the ends placed in the microflame of the Bunsen burner for three or four seconds. The tube was rotated in the flame to avoid tapering or bulb formation at the end of the tube.

TABLE I.

Results of Hæmatocrit Estimations of Capillary Blood and Venous Blood, with the Micro and Wintrobe Methods.

Case Number.	Capillary Hæmatocrit: Micromethod.	Venous Hæmatocrit.	
		Micromethod.	Wintrobe (Uncorrected).
1	40·5	39·0	40·5
	39·5	38·5	41·0
	39·5		
2	44·0	42·5	44·5
	43·5	42·5	44·5
	43·5	42·5	
3	37·0	37·5	38·0
	36·5	37·5	38·0
		37·5	
4	33·0	33·5	35·0
	33·0	34·0	35·0
	33·0	34·5	
5	46·0	44·0	46·0
	46·5	44·5	46·0
	46·0	44·5	
6	43·0	41·0	44·0
	42·5	41·5	43·5
	42·0	41·0	
7	43·5	43·5	44·0
	44·0	43·5	43·5
		44·0	
8	41·0	41·0	43·0
	41·0	41·0	43·0
		41·0	
9	40·5	41·0	42·0
	40·0	40·5	41·5
	39·5	40·5	
10	44·0	41·5	43·5
	44·0	41·5	43·5
	44·0	42·0	
11	32·5	31·5	32·5
	33·5	32·5	32·5
		32·0	
12	37·5	37·5	38·0
	37·5	37·5	38·0
	38·0	37·0	
13	43·5	43·5	45·0
	43·5	43·5	45·0
	43·0	43·5	
14	38·0	38·0	39·5
	37·5	37·5	39·5
	37·5		
15	42·0	41·0	42·0
	41·5	41·5	42·5
	41·0	41·0	
16	39·5	39·0	40·0
	39·5	38·5	40·0
	39·0	39·0	
17	39·5	39·5	40·0
	40·5	39·5	40·0
	40·0	38·5	
18	43·5	40·5	42·0
	43·5	40·5	42·0
		41·0	
19	43·0	41·5	43·0
	43·0	42·0	43·0
	42·5	41·5	
20	41·5	42·0	43·5
	42·5	41·5	43·0

The tubes were placed in the head of the centrifuge with the sealed ends touching the rim of the head, the cover was replaced and the blood was centrifuged for five minutes. The hæmatocrit was read immediately as described.

The Wintrobe tubes were spun for 30 minutes at 3000 r.p.m. with a radius of 15 cm. No corrections have been made in the results for trapped plasma.

Results.

The results of the hæmatocrit estimations are presented in Table I. This shows the reproducibility of the results with repeated tests and the close correlation between capillary and venous blood. Similar results were obtained in an earlier series of 30 patients when technical difficulties with the collection and reading of the blood had been overcome.

In this series, the hæmatocrit of the capillary blood was in most cases slightly higher than that obtained with the venous blood. With the exception of Case 18, the maximum difference of packed cell volume of capillary blood when compared with that of venous blood by either the micro method or the Wintrobe method was less than 2·2%. In this case the patient had cold hands, and the high capillary hæmatocrit is probably due to undue compression of the finger during collection.

Discussion.

The micromethod for the determination of the hæmatocrit of blood has been shown by Biggs and MacMillan (1948) McInroy (1955) and McGovern *et alii* (1955) to have the same degree of accuracy with repeated estimations as the established Wintrobe method. This was confirmed in this series.

If corrections are made for trapped plasma with the Wintrobe method and complete packing is assumed with the micromethod, the agreement between the two methods is close.

Hæmoconcentration or hæmodilution can be produced by excessive manipulations in the collection of capillary blood; but Wintrobe (1956) has made the following statement:

> It has been repeatedly shown that there is no difference in counts made on venous blood as compared with capillary blood, if proper precautions to secure a freely flowing sample are observed in each instance.

With this method, the blood is packed in less than five minutes with a force of 12,000 G. Experiments have shown that this is about the maximal centrifugal force permissible if hæmolysis is to be avoided. The technique of collection of the samples, sealing of the tubes and reading is readily mastered, and consistent results are obtainable.

This method has considerable advantages, as only small amounts of capillary blood are necessary, the method is reliable and it can be applied in all cases. Care is necessary in the interpretation of results when a freely flowing sample is not obtained, as in cases of shock.

Considerable time is saved with the method, not only in centrifugation, but also because the use of disposable capillary tubes eliminates the time required for cleaning.

Now that this machine has become available, together with the reader and capillary tubes, which are of uniform bore and can be discarded after use, this method should come into more general use.

Summary.

The use of the microhæmatocrit centrifuge and reader in an obstetric and gynæcological unit is described.

With this machine, the packed cell volume can be determined with capillary blood. The method produces complete packing in five minutes in disposable, capillary tubes.

The method is of particular use in the treatment of infants requiring full blood investigations, but can, with benefit, be applied more generally.

Acknowledgements.

This work was carried out during the tenure of the Fotheringham Research Fellowship of the Royal College of Obstetricians and Gynæcologists for 1958. I should like to thank Professor Bruce T. Mayes for the interest he has taken in the project, and Dr. John Greenwell for his helpful support at the Royal Hospital for Women, Sydney, where the work was carried out.

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Reviews.

Cortisone Therapy: Mainly Applied to the Rheumatic Diseases. By J. H. Glyn, M.A. (Cantab.), M.D., M.R.C.P., D.Phys.Med., with a foreword by The Rt. Hon. The Lord Cohen of Birkenhead, M.D., D.Sc., LL.D., F.R.C.P., F.A.C.P.; 1957. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 172, with four illustrations. Price: 21s. (English).

DR. GLYN is Consultant in Physical Medicine to the Prince of Wales and Tottenham group of hospitals. He has worked in close collaboration with specialists in the rheumatic diseases in both the United Kingdom and the United States of America, during clinical trials with cortisone and related steroids. It is obvious from reading his book that he has had a very wide experience in the practical management of patients with the various forms of chronic joint disease and collagen diseases.

In the past eight years many papers have been published about the use of the steroids, offering readers a large number of observations from which it has often been difficult to draw valid conclusions. The results of clinical trials such as those conducted by the Medical Research Council in Great Britain have been of great value because of the careful statistical control but the selection of cases has been rigid and somewhat limited. We are therefore indebted to Dr. Glyn for this excellent monograph, in which he covers in a relatively brief compass the historical, theoretical and practical aspects of therapy with cortisone and related steroids. Although more attention is given to the treatment of rheumatoid arthritis than to other conditions, the general indications for steroid therapy, both local and systemic, are covered. There is a very strong emphasis on the practical aspects of management, with very detailed instructions which should guide even the inexperienced practitioner along the rather difficult way of steroid therapy.

The whole question of the propriety of using steroids for non-fatal conditions has engendered heated arguments. Dr. Glyn presents the arguments for and against in a balanced and sensible manner which will probably stand the test of time.

The section dealing with the intraarticular and local use of hydrocortisone is particularly good. There is a valuable appendix by J. G. Bearn on the anatomy and technique of intraarticular and soft-tissue injections, and another on the assessment of clinical progress by functional tests and activities of daily living, with a suggested chart for use in short-term trials.

This book is an extremely valuable contribution to the literature on the subject, and is recommended for all who may use steroid therapy.

Neomycin: Its Nature and Practical Application. Edited by Selman A. Waksman; 1958. Published for the Institute of Microbiology by The Williams and Wilkins Company, Baltimore. 9" × 5½", pp. 414. Price: 55s.

NEOMYCIN may well be considered in the therapy of many infections, and this is a complete reference book in the use of that drug. The property that severely limits the usefulness of neomycin is its unhappy tendency to cause disturbances of kidney function and loss of hearing. They occur as the result of repeated parenteral administration, and are rarely seen with oral administration and then only after long-continued use of high doses.

The particular value of neomycin is in the form of topical preparations for use on the skin, in the nose and on the conjunctiva. It is available in many commercial preparations in ointment form, and is often combined with hydrocortisone or one of the other topically used antibiotics.

Neomycin exhibits rapid bactericidal action, which explains some of the dramatic clinical results described in this book. Organisms do not show cross resistance with penicillin and the broad-spectrum antibiotics. Organisms resistant to neomycin are likely to be resistant to streptomycin, but the reverse is not necessarily the case.

Diarrhœal disease is one of the most common diseases affecting mankind, and it was hoped that neomycin would be of some value in therapy. The reports in this volume indicate its usefulness in the therapy of *Escherichia coli* infections, which have been frequently reported abroad as a cause of diarrhœa in infants. However, it is pointed out that clinical value of an antibiotic in diarrhœal diseases is difficult to assess, and that fluid replacement to the patient is more important than antibiotic therapy.

Neomycin is probably the most efficient orally given antibiotic for reducing or eliminating bowel organisms before open operation on the intestinal tract. It is considered by some surgeons, however, that reduction in bowel organisms is unnecessary for satisfactory results. One of the risks to patients undergoing bowel surgery is "super-infection" with resistant staphylococci and the development of membranous enterocolitis. Even if neomycin cannot prevent this, it can well be considered for its therapy.

By his discoveries and the development of streptomycin and neomycin, Selman A. Waksman has contributed greatly in the field of antibiotics, and this volume edited by him will enable clinicians to add to their knowledge of that subject.

Rehabilitation After Illness and Accident. *Edited by Thomas* M. Ling, M.D., M.R.C.P., and C. J. S. O'Malley, C.B.E., M.B.; 1958. London: Baillière, Tindall and Cox. 8½" × 5½", pp. 126. Price: 12s. 6d. (English).

IN the compilation of this short series of essays on various aspects of rehabilitation the editors have been assisted by six colleagues. This has resulted in some repetition, but has made each contribution complete in itself. It is not surprising that a good deal of emphasis is placed on the conditions prevailing in the Welfare State and their influence on rehabilitation of the sick and injured. Factors of importance include the effect of sick pay on the duration of incapacity, the complex legislative and administrative machinery involved in the later stages of resettlement and the still important role of voluntary agencies.

Mention is made of the Piercy Committee, set up jointly in 1953 by the Ministries of Labour and Health. This reported in 1956 that rehabilitation is a single process in which the emphasis at the beginning is on medical treatment and at the end on work, with scope for development on both the hospital and the industrial side. It is thought that consultants are still slow to consider the rehabilitation needs of their patients, and that further education of the medical profession, including medical students, is still needed. General practitioners should take more responsibility for rehabilitation, backed up by a greater awareness of its potentialities.

Social and emotional factors in both patient and relatives are adequately covered in several parts of the book. There is a changing pattern of environment for the patient in hospital under the care of nurses and doctors, in the home as a convalescent and in the rehabilitation centre or industrial rehabilitation unit (operated in England by the Ministry of Labour). Often the stage of dependency of an accepted illness may persist too long. As the objective of treatment should be the placement of the patient in employment as soon as possible, the personality of the person treating the patient at each stage of his illness is important. At the stage of recovery of function, it is more important to do things with patients than to do things to patients.

From the industrial viewpoint, mention is made of the compulsory employment of a quota of 3% of disabled in places employing over 20 persons, of special rehabilitation workshops in some large concerns, and of the contribution made by Remploy factories in the provision of sheltered employment for the severely disabled.

Special chapters are devoted to the rehabilitation problems of certain conditions, such as thoracic disease, cerebral surgery and injury and general and orthopædic surgery. There is a good section on occupational therapy, which gives

rather more detail about the specific uses of this form of treatment than is found in the other chapters relating to general treatment. In fact, one might criticize much of the material as being too general and too diffuse. In general, however, the book is a good general review of the purpose and use of rehabilitation techniques as applied in England.

Metabolic Disturbances in Clinical Medicine. Edited by G. A. Smart, B.Sc., M.D., F.R.C.P.; 1958. London: J. and A. Churchill, Limited. 9" x 5½", pp. 368, with 35 illustrations. Price: 45s. (English).

THIS is a distinctly useful book written by nine British and five American contributors under the general editorship of Professor Smart. Its aim is somewhat different from that of other recently reviewed works dealing with metabolic abnormalities essentially from a basic viewpoint; it describes for the practising clinician certain disturbances of metabolism likely to be encountered in his work. The contributors are a new group, including some well-known names, and show their individual style to some extent in their respective sections.

The chapters include topical subjects, such as aldosterone and aldosteronism, œdema in chronic congestive heart failure and atherosclerosis, as well as dealing specifically with metabolic aspects of renal, liver and bone diseases, etc. The effects of metabolic disturbances upon the cardiovascular, hæmatopoietic and central nervous systems are also discussed. A chapter dealing with metabolic disturbances following injury is a welcome inclusion, likewise one on oxygen therapy. An initial chapter dealing with general nutrition in clinical medicine is a worthwhile introduction, although it could justifiably have been more discursive. A number of the chapters suggest an undergraduate emphasis in part; this is not a significant criticism, although some sections may thus prove disappointing to the more widely-read. However, the book fulfils its purpose, and should help both the undergraduate and the graduate to develop perspective. Naturally, as has been pointed out before, these volumes have to limit their subjects if they are to remain practical; as this book is labelled "first edition", perhaps other aspects of clinical medicine will be discussed in subsequent editions.

Selected references are given either at the end of the chapters, or in their sections. The index is reasonable, but the longer chapters could benefit by an initial table of contents. The volume, which has a reasonably wide coverage, has much to recommend it, but it is probably unfortunately priced.

Progress in Arthritis. Edited by John H. Talbott, M.D., and L. Maxwell Lockie, M.D.; 1958. New York and London: Grune and Stratton. 9" x 5½", pp. 464, with many illustrations. Price: $12.50.

THIS book is a collection of 27 papers written by recognized American authorities on selected subjects in the broad field of arthritis. Research, both basic and clinical, is being actively pursued in the United States at the present time in the field of the arthritic diseases. Congress alone, through the National Institute of Arthritis and Metabolic Diseases, is devoting an annual sum of about $20,000,000, while the American Arthritis and Rheumatism Foundation and various private individuals and foundations, as well as the various pharmaceutical companies, probably provide even greater sums for research and training. As a consequence, tremendous advances have been made in the understanding of this very difficult group of diseases, even though the ultimate goal has so far remained elusive. The editors themselves have made significant contributions—Dr. Lockie in the field of rheumatoid arthritis and Dr. Talbott in the metabolic and clinical aspects of gout.

The production is of high quality, the book is printed on art paper and has numerous illustrations, some in colour. A comprehensive list of references is included at the end of each contribution. These include a substantial number of British and other non-American papers. It must be difficult to decide what to include and what to omit from a book of this type. The editors have included in various ways rheumatic fever, rheumatoid arthritis, osteoarthritis, gout, ankylosing spondylitis, Dupuytren's contracture and the shoulder-hand syndrome. Apart from a general paper on the management of rheumatoid arthritis, special papers on phenylbutazone and steroids are included. There is special mention of rehabilitation. Other titles include "Fibrin-Like Substances in Collagen-Vascular Disease", "The L-E Cell Phenomenon" and "Serological Reactions in Rheumatoid Arthritis", and three papers deal respectively with the heart, the nervous system and the joints in connective tissue diseases.

One might perhaps criticize certain views expressed; but the editors themselves state that they do not necessarily agree with everything in the book, responsibility for which is borne by the individual contributors. However, we in this country cannot let pass unchallenged the statement that the reported sex ratio of six males to four females for ankylosing spondylitis (Parr, White and Shipton) in Australia, which is at variance with the usually accepted figure of about nine males to one female, is due to the predominantly female Australian population.

One might criticize also the arrangement of the contents, which does not follow a completely logical pattern. In general, however, there is in this book, collected in a relatively small compass, a well-written review of many of the advances in our knowledge of the arthritic diseases, together with a good bibliography for more intensive study of source material for anyone interested in this field.

The Kidney: An Outline of Normal and Abnormal Structure and Function. By H. E. de Wardener, M.B.E., M.D., F.R.C.P.; 1958. London: J. and A. Churchill, Limited. 9½" x 5½", pp. 348, with 74 illustrations. Price: 45s. (English).

THE wide range of subject matter presented in this book is a measure of the great increase in and reorientation of the knowledge of renal disease that has developed over the past ten or fifteen years. Its publication is timely. As well as providing a complete outline of renal disease suitable for students (as the author intends), it must be a very useful refresher for those in practice.

Brevity is the keynote; many chapters consist of one or two pages and few of more than 10; yet the book deals with the widest range of renal disorders, and there are few aspects of the subject which are not touched upon. This means that there is little room for discussion, for the presentation of evidence or for the development of theory, and the style is therefore frankly didactic. It is the greatest pity, however, that there are no references through the text; the bibliography at the end of a chapter is a poor substitute.

Preliminary chapters on anatomy and physiology are followed by consideration of a group of syndromes of disordered function, including the nephrotic syndrome, acute renal failure, chronic renal failure and, interestingly, the acute nephritic syndrome, which covers the various types of collagen disease, anaphylactoid purpura and radiation nephritis as well as glomerulo-nephritis. Then follows a series of many chapters on specific diseases or clinical states. Some of the more interesting include radiation nephritis, renal vein thrombosis, porphyria and innate renal tubular defects. The merits of brevity notwithstanding, some important matters receive perhaps too little attention. These include the concept of osmotic diuresis as the mechanism of isosthenuria in chronic renal disease, modern views on the process of concentrating urine and the role of renal artery obstruction in hypertension, recently the subject of some important papers.

Throughout the book, many practical procedures and tests of renal function are described clearly and in detail. Indeed, the emphasis of the work is directed towards the clinical aspects of renal disease, and the author obviously writes from his own experience, quoting many of his own original investigations.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Clinical Introduction to Heart Disease", by Crighton Bramwell, M.A., M.D., F.R.C.P.; 1959. London, New York and Toronto: Oxford University Press. 8½" x 5½", pp. 240, with 61 illustrations. Price: 39s. 3d.

Written specially for general practitioners and senior medical students.

"Plague Fighter: The Autobiography of a Modern Chinese Physician", by Wu Lien-Teh, M.A., M.D. (Cantab.), Mast.P.H., Litt.D., Sc.D. (St. Johns, Shanghai), LL.D. (Hongkong). 1959. Cambridge: W. Heffer & Sons, Limited. 8½" x 6", pp. 678, with illustrations. Price: 30s. (Abroad).

A book of memoirs.

The Medical Journal of Australia

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AUSTRALIAN SCIENCE AND INDUSTRY IN THE WAR OF 1939-1945.

In the war of 1914-1918 Australia supplied her troops with food and clothing, rifles and ammunition, but her dependence on outside sources for armament and high explosives emphasized the inadequacy of the existing defence scheme. The great development of secondary industry and of scientific organizations in the intervening years, largely as a result of lessons learnt in the earlier war, enabled Australia in the war of 1939-1945 to meet a challenge for mass production of materials and articles of a higher degree of complexity and accuracy than had hitherto been attempted here. After 1939 most industries became increasingly involved in munition work under the direction of Essington Lewis, and munition laboratories were fully engaged. The Council for Scientific and Industrial Research suspended all work not bearing directly on the war effort, but there was great concern amongst scientists generally because of the lack of any definite scheme to organize scientific skill and experience. However, constant pressure by the Australian National Research Council induced a reluctant Government at the end of 1942 to establish a Scientific Liaison Bureau under Professor E. Ashby, which thereafter functioned admirably as a coordinating body. Its work on tropic-proofing alone amply justified its existence. In a recent volume in the series on Australia in the war of 1939-1945, Professor D. P. Mellor[1] expresses the opinion that the efficiency of the Bureau was greatly reduced because it was not given the support and status which it deserved. Concerned mainly with armament, Professor Mellor's history deals also with the building and repair of aircraft and ships, with radar and communications, with meteorology, camouflage and tropic-proofing, and with other problems which became increasingly important as the area of conflict extended. It is a valuable record of a most important phase in the history of Australian science and industry. It is written primarily for the layman, but its range is wide, and there will be few readers who do not appreciate, in regard to some of the sections at least, the explanation of the technical problems involved and how they were solved. It has entailed much research and contains a wealth of material not readily available elsewhere.

The question of drugs and fine chemicals, already fully dealt with by the medical historian, is here approached from rather a different angle. Full acknowledgement is given to the outstanding work of Alan Newton and the Medical Equipment Control Committee, and stress is also laid on the valuable contribution of the Drugs Sub-Committee of the Australian Association of Scientific Workers, who carried out laboratory work essential for the synthesis of various drugs for which raw materials were available; many of these drugs were of the utmost importance. The value of the anti-malarial work carried out by the Research Unit at Cairns, and the taxonomic work of various entomologists which facilitated the recognition of specific vectors, are fully appreciated, as also are the efforts of the Commonwealth Serum Laboratories, particularly their achievement in making available to the Army locally made penicillin as early as 1944. There are other medical references elsewhere in the history, notably to the "aerodynamic anti-G suit" devised by Professor F. S. Cotton for control of blackouts, and to the occurrence of industrial diseases in ammunition factories.

The problems of food and nutrition are considered in detail. After the entry of Japan into the war, Australia was committed for a time to supplying the needs of all the Allied Forces in the South-West Pacific area in addition to feeding her own population. This produced manifold problems, many of them unforeseen, and involved enormous increases both in production and in processing, the achievement of which was in no small measure due to supervision and advice from skilled American executives and technicians. There were remarkable improvements in the rate and quality of can manufacture, and food technology generally received an extraordinary stimulus, the effect of which is still evident. Storage of food was a difficult problem, particularly in the tropics, and canned food especially suffered from lack of tropic-proofing. Dehydrated food produced palatable meals, but it was never very popular and required careful storage. There still remained the problem of serving food to the soldier in the field in an attractive palatable form and avoiding monotony to minimize wastage; in this regard there is special commendation of the Wiles steam cooker. The following conclusions are reached: (*a*) that despite difficulties of transport and storage, servicemen were surprisingly well fed; (*b*) that more food was produced and less wasted, and that fewer Australians went hungry than at any other time in the country's history (there are probably some people, both service and civilian, who would not be in entire agreement); (*c*) that Australia provided more food per head of population to the Allied larder than did any other country.

Not every project undertaken was an unqualified success. The manufacture of optical munitions was an example of splendid cooperation between science and industry which achieved very satisfactory results, but in some instances much time and money were expended on enterprises which made no contribution to the war effort. At times organizations or individuals were allotted tasks that were beyond their capacity, or, before a project was completed, some change of policy had made it superfluous. The making of torpedoes was

[1] "Australia in the War of 1939-1945. Series 4 (Civil): The Role of Science and Industry", by D. P. Mellor; 1958. Sydney: Angus and Robertson, Limited. Canberra: Australian War Memorial. 9¼" x 6", pp. 750, with illustrations. Price: 30s.

regarded as one of the outstanding examples of precision engineering undertaken by the Commonwealth, yet for various reasons no Australian-made torpedo was ever used in action. However, such projects, even if they did not augment the allied military strength, did make a definite contribution to Australia's strength as an industrial nation. Indeed, it seems clear from this account that industry derived many benefits from its experience in the war effort. There was a great development in the variety and complexity of manufacture, from which their staffs could benefit, and which provided full scope for their resourcefulness and ability to improvise. This ensured higher and more accurate standards in mass production and prepared for the remarkable post-war expansion of secondary industry. From our own particular point of view, scientific and medical, it may well be pointed out that the contribution of science to the war effort has produced a much wider and more realistic appreciation of the scientist and his work in the modern world: scientific education and research are being fostered and encouraged as never before with a realization—stimulating, not defeatist—that we lag behind in the race.

Current Comment.

THE STONE.

THE stone which troubled our ancestors in the time of Pepys was notoriously the vesical calculus; its manifestations were urgent and its cure was dramatic. With improved standards of nutrition and medical care the incidence of vesical calculus has, in Europeanized countries, greatly diminished in frequency, but its bedfellow the renal calculus is still common, and in view of the diversity of the factors which may cause its formation, is likely to remain so. In a recent review, R. A. Melick and P. H. Henneman[1] summarize the data from the records of all patients attending the Stone Clinic of the Massachusetts General Hospital in the five years to December, 1954. The total number of cases considered was 207. Chemical analysis of the stones was performed in all but 52 cases. Mixed calcium phosphate and calcium oxalate stones were the commonest variety, and were found in 47 patients. Other types found were: calcium oxalate (42), calcium phosphate (32), uric acid (20), cystine (4), and phosphate stones containing calcium, magnesium and ammonium (23). Fourteen patients had other types of mixed calculi. In an attempt to establish the reason for stone formation all patients were carefully investigated. In 68 cases stone formation was considered to be due to hypercalciuria, which was caused by a number of factors. Twenty of these patients suffered from hyperparathyroidism, and had undergone surgical treatment on that account (calculus formation was apparently the presenting symptom of the disease). In 13, hypercalciuria was believed to be due to demineralization of the skeleton from prolonged bed rest or other cause. In 11 patients it was attributed to excessive intake of milk (more than two quarts per day), alkalis or vitamin D over long periods. Other causes of hypercalciuria were renal tubular acidosis (six patients) and idiopathic hypercalciuria (18 patients). Patients in the last group had a urinary calcium excretion of over 200 mg. in 24 hours; 15 of them were males, and five had a family history of stone.

Stone formation not associated with hypercalciuria embraced a number of other conditions. Six patients had cystinuria, and, as would be expected in this familial condition, most of them gave a history of stone in other members of their family; cystine stones, it may be noted, are always seen on the X-ray film because of their sulphur content. The uric acid stones were found to be most commonly associated with acid urine, rather than a raised excretion of uric acid; these stones are not radio-opaque unless secondary infection has led to the deposit of phosphates. In some members of this group stone formation was associated with gout, polycythæmia or persistent diarrhœa (three patients with ileostomy or colonic resection), but in the majority the condition was idiopathic, the only abnormality being a persistently acid urine. It is an interesting fact that 13 of the 14 patients in this last category were of Italian or Jewish origin.

Stones composed of calcium phosphate with magnesium ammonium phosphate were found in 34 patients, and were considered to be the secondary result of urinary infection; however, in only three of these cases did the secondary infection appear to be the primary cause of the stone; in the remainder the infection was itself secondary to stone formation. Finally, there was a group of 31 patients with more or less pure calcium oxalate stones, in whom no obvious cause for stone formation could be discovered. These stones were usually small, and were often passed spontaneously; possibly as a result of the low rate of instrumentation in this group, the incidence of infection was also low. In 77 other patients with stones of various sorts, no cause could be found to account for the condition.

Other points investigated by Melick and Henneman included the age of onset, which was found to differ considerably between groups. They did not find any evidence that mechanical or anatomical defects were a factor in the ætiology of renal stone formation, though they admit the possibility that such defects could be a causative factor, and state that they should be excluded in all cases. In the most recent follow-up of these patients, it was found that 109 were free of stones and 155 were free of infection. Melick and Henneman conclude that a cause for stone formation can be found in at least half of all patients with renal stones, and that combined medico-urological management will render most patients free from stones and infection.

This comment would be incomplete without reference to a paper by H. S. H. Wardlaw, which appeared in this Journal in 1952.[1] In it Wardlaw presents an analysis of the location, age incidence and chemical composition of urinary stones of all sorts, collected at Sydney Hospital between 1925 and 1950, and an analysis of the location and age incidence of all stones whose presence was diagnosed in Sydney Hospital patients between 1942 and 1950. In the latter group many of the stones were not available for analysis. This paper gives an interesting picture of the age and sex incidence of urinary calculi, in their several categories, including renal, ureteric, vesical and urethral stones, in an Australian population, but makes no attempt to study the causes of stone formation in the patients concerned.

GROWTH AND GROWTH HORMONE.

ONE of the most distressing situations in clinical endocrinology lies in the absence of any treatment for pituitary dwarfism. The congenital failure of growth hormone secretion does not manifest itself for several years after birth, because growth continues at a normal rate during this time, even in the absence of growth hormone. Eventually, it gradually dawns upon the parents that their child is shorter than his fellows. Commonly, those affected are of normal intelligence, and their playmates show no regard for the painful dilemma in which the sufferers find themselves. Mothers of such boys become unduly solicitous, and a sense of guilt leads them to search in vain for a cure; but there is no peace for these sensitive children, of whom some become recluses, while others over-compensate.

Our understanding of growth is so limited that no universally accepted definition has been suggested. To the lay mind, growth usually signifies increase in height,

[1] *New Engl. J. Med.*, 1958, 259: 307 (August 14).

[1] MED. J. AUST., 1952, 1: 180 (February 9).

and from a clinical point of view this is the most urgent feature of growth. Physiologists have defined growth as the laying down of new tissue, and the essential problem of laying down new tissue is the synthesis of protein; this is reflected by nitrogen retention. Growth hormone has been shown, in animals, to cause nitrogen retention, the retained nitrogen being used in the synthesis of new protein. These changes are reflected by a fall in blood amino acid and non-protein nitrogen levels. Growth hormone further stimulates the mobilization of depot fat, enhances its oxidation in the liver and also possibly opposes the hepatic synthesis of fat.

F. G. Young, in the Sydney Ringer Lecture for 1950,[3] has summarized these changes by pointing out that growth hormone leads to a high body content of protein, water and minerals together with a low content of fat—that is, the changes seen during the growth of young animals. During periods of active growth, the intake of food is relatively high in proportion to total body weight. In addition, Young has shown that growth hormone is diabetogenic in fully grown animals, especially in dogs and other carnivorous species. In other words, the hormone produces growth during "puppyhood" and diabetes during adulthood. The site of this action is uncertain, but it seems likely that growth hormone opposes the action of insulin (perhaps interfering with its action upon hexokinase[3]) and in this way causes hyperglycæmia. Eventually, hyperglycæmia causes degenerative changes in the beta cells of the pancreatic islets, and these may prove permanent. Absence of growth hormone appears to be largely responsible for the amelioration in pancreatic diabetes following hypophysectomy (Houssay effect).

It was understandably disappointing to discover that growth hormone was species-specific. It is certainly true that smaller peptide fractions of the whole molecule are capable of producing growth; this has suggested that the whole molecule contains a nucleus responsible for growth, and that this is modified by other amino acids which cause species-specificity. In spite of these discoveries, chemical changes in the structure of animal growth hormone have not made such preparations active in man. The announcement by workers in Boston that growth hormone extracted from the human adenohypophyses at autopsy (called H.G.H.) was active in man naturally excited great interest. The original claims have recently been substantiated by a sober report from the Medical Research Council.[4]

In a preliminary report, M. S. Raben[4] states that H.G.H. was not found to possess any measurable amount of other pituitary hormone activity, even when large doses were given. No gonadotrophic activity was found. In a pituitary dwarf, he recorded a rise in the serum content of inorganic phosphate and alkaline phosphatase to levels characteristic of growing children. No allergic phenomena, local or general, were encountered. Therapy was given continuously during a period of ten months by intramuscular injection, and during the last seven of these months the dose was 2 mg. three times weekly. The rate of growth in previous years had averaged 0·5 inch per year, but during the ten months of treatment the rate was 2·6 inches, which is slightly greater than the rate of growth of a normal boy of the same height. The period of growth was associated with a rise in food intake.

The Medical Research Council report begins with an account of the tedious procedure used for extraction and the elaborate tests used to check the purity and the absence of toxicity in the final product. One pituitary gland yields approximately 1 mg. of H.G.H. Metabolic studies were conducted with the use of controls and took place in two stages: the first involved a total dose of 10 mg. of H.G.H. per patient and the second a total of 30 mg. per patient. Nitrogen retention and a fall in blood urea and urine nitrogen levels were seen. Retention of potassium, sodium, magnesium and phosphorus was recorded in amounts expected if the retained nitrogen was devoted to protein synthesis, except in the case of sodium, when retention was excessive in relation to protein synthesis. A slight rise in fasting blood sugar content was also noticed. These workers found an increase in urinary calcium content, which they suggested could be an over-dosage effect. The effect of a single injection of H.G.H. lasts for several days, and the authors suggest that weekly or twice-weekly injections may represent the most economical and effective method of administration.

It is worth mentioning that with the possibility that active H.G.H. will soon become available, pituitary dwarfs who also show failure of sex development should be given H.G.H. before they are given gonadotrophins, because of the possibility that stimulation of the gonads will hasten the closure of epiphyses and hence limit the extent of increase in height when growth hormone is used. Finally, it is to be hoped that further reports concerning the availability of H.G.H. will not be made in the lay Press before supplies are at hand. The process of preparation is necessarily lengthy, and experience with the hormone is extremely limited as yet; so that the hopes of anxious parents should not be prematurely raised.

[1] *Brit. med. J.*, 1951, 2:1167 (November 17).
[2] Med. J. Aust., 1959, 1:437 (March 28).
[3] *Lancet*, 1:7, 1959 (January 3).
[4] *J. clin. Endocr.*, 18:901, 1958.

FOOD ADDITIVES.

THE term "food additives" is used to refer to non-nutritive substances added intentionally to food, generally in small quantities, to improve its appearance, flavour, texture or storage properties. In countries with a high standard of living, the use of these substances makes it possible to provide exacting consumers with a wide selection of exotic or seasonal products throughout the year and to give foods an attractive appearance. In many countries, especially those in tropical or subtropical regions where, in some cases, modern storage facilities are lacking, food additives may help to prevent spoiling of seasonal surpluses. Thus they are of great economic value, but unfortunately they are not free from danger. On the one hand they enable unscrupulous producers to conceal defective merchandise; on the other they may be toxic for the consumer or reduce the nutritional value of the food to which they are added. The importance is therefore obvious of a recently issued report of the Joint FAO/WHO Expert Committee on Food Additives,[1] which is intended to give persons interested in the question of food additives on scientific or administrative grounds a general picture of the type of information which should be available about any additive before its use in food is officially approved. The report provides a list of publications on the subject which have already appeared and describes briefly tests suitable for the study of acute, short-term or long-term toxicity, taking into account the part that such tests may play in the determination of harmlessness. It is pointed out that an adequate knowledge of metabolic and biochemical effects of a food additive may provide in some cases a satisfactory background for recommendations on safety for use, and some biochemical aspects that may be profitably studied are listed. A number of recommendations are made to FAO and WHO in relation to the use of food additives and especially to the problem of the possible carcinogenic and mutogenic action of such substances. The expert committee considers that the problems of chemical carcinogenesis and mutogenic action, which are not confined to food additives, are important enough to merit further consideration at a later date by a group having amongst its membership a number of appropriately qualified workers in the field of cancer research. Meantime there would be general agreement with the committee's opinion that no proved carcinogen should be considered suitable for use as a food additive in any amount.

[1] "Procedures for the Testing of Intentional Food Additives to Establish Their Safety for Use", World Health Organization Technical Report Series No. 144; 1958. Geneva: World Health Organization. 9½" × 6¼", pp. 20. Price: 1s. 9d.

Abstracts from Medical Literature.

MEDICINE.

Chlorpromazine Jaundice.

W. F. JEBHART *et alii* (*A.M.A. Arch. intern. Med.*, June, 1958) present an analysis of 20 patients with chlorpromazine jaundice, in 15 of whom liver biopsies were undertaken. All of the patients were jaundiced, 16 had pruritus, 15 had hepatomegaly with tenderness, 14 had an influenza-like syndrome, 11 had abdominal pain, and eight showed eosinophilia. Duration of administration of the drug ranged from two to 58 days, but one patient received only 75 mg. daily for two days. Jaundice usually developed from two to four weeks after the initial administration of the drug. Liver function tests consistently showed the pattern of an obstructive jaundice rather than of hepatocellular disease, and the serum alkaline phosphate level, which seems to be the best test for this type of jaundice, was elevated in all 20 patients. Serum levels of cholesterol and cholesterol esters were elevated in each of 12 cases in which these tests were done. The results of flocculation and turbidity tests were uniformly normal, as were the serum protein values. The duration of jaundice ranged from eight days to 10 months. In two cases in which the patient was pregnant, neither the pregnancy nor the fœtus was adversely affected. Follow-up studies of all patients in this group indicated eventual recovery without any residual liver damage. The findings on needle biopsy indicated an absence of necrosis of any type within the liver cells; this usually served to differentiate chlorpromazine jaundice from viral hepatitis. The absence of large accumulations of bile, either as lakes or within larger ducts, serves to differentiate chlorpromazine jaundice from that due to extrahepatic or large bile duct obstruction. However, the authors conclude that the hepatic lesion produced by chlorpromazine cannot be morphologically distinguished from other drug-induced lesions, such as those induced by methyltestosterone ingestion. Although no effective treatment is proposed, a "hepatitis regimen" is advocated for these patients, with a high-carbohydrate, high-protein diet, bed rest, and supplementary vitamins. Steroid therapy was found ineffective in five patients.

Paroxysmal Supraventricular Tachycardia.

M. R. HEJTMANCIK, G. R. HERRMANN AND J. C. WRIGHT (*Amer. Heart J.*, November, 1958) have studied 175 consecutive patients with paroxysmal supraventricular tachycardia seen at the University of Texas Hospital. They remark that paroxysmal supraventricular tachycardia has a reputation of being a benign, well-tolerated arrhythmia occurring usually in otherwise normal individuals. However, it had been their clinical impression that such a disorder also commonly occurs in patients with diseased hearts. In their material they found that 75% of their patients, usually the older age group, had evidence of heart disease, most commonly hypertensive or ischæmic. Serious clinical symptoms were rarely observed in patients with normal hearts, but chest pain, heart failure and shock were frequent complications when the disorder accompanied organic heart disease. The usual method of carotid sinus pressure and other means of vagal stimulation were much less effective in patients with organic heart disease than in normal patients. The authors recommend the use of "Prostigmin" 0·5 mg. injected intramuscularly. If this also failed, digitalis therapy was found to be the most effective, provided that digitalis was not a factor in the production of the arrhythmia. Paroxysmal supraventricular tachycardia with atrio-ventricular block was usually a manifestation of digitalis intoxication. Gratifying restoration of sinus rhythm usually followed upon discontinuation of digitalis and the administration of potassium salts.

The Electrocardiogram in Scleroderma.

J. ESCUDERO AND E. MCDEVITT (*Amer. Heart J.*, December, 1958) analyse the electrocardiographic changes in 60 cases of scleroderma. The patients fell into two groups, group I (31 patients) with typical scleroderma of the skin without evidence of visceral involvement, group II (29 patients) with typical scleroderma of the skin and visceral involvement of heart, lungs, kidneys, œsophagus, etc. In group I the authors found electrocardiographic abnormality in 25%, in group II the electrocardiogram was abnormal in over 75% of cases. The most frequent alteration in all cases was notching of the *P* wave in standard leads. Because of the presence of auricular disease in those cases in which autopsy was performed, the authors consider that this finding creates a suspicion of myocardial damage by scleroderma.

A Paradoxical Manifestation of Digitalis Intoxication.

M. BACANER (*Amer. Heart J.*, November, 1958) discusses the occurrence of a rapid ventricular rate during atrial fibrillation as a paradoxical manifestation of digitalis intoxication. Although it is well known that during atrial fibrillation digitalis overdosage may excessively decrease ventricular rate, it is not generally recognized that digitalis intoxication may occasionally be manifest by a marked increase in ventricular rate. The author describes three patients with long-standing atrial fibrillation all taking large doses of digitalis. In each instance an increase in ventricular rate followed recent administration of a mercurial diuretic and was thought to be associated with loss of potassium during diuresis. Intravenous administration of potassium salts resulted in prompt and progressive slowing of the ventricular rate over a two-hour period.

The Aortic Lesion of Ankylosing Spondylitis.

B. M. ANSELL, E. G. L. BYWATERS AND I. DONIACH (*Brit. Heart J.*, October, 1958) report the cases of two patients with aortic lesions and aortic incompetence associated with ankylosing spondylitis. The authors agree with Walter Bauer that this is a specific lesion related in some way to the connective tissue changes in ankylosing spondylitis. Histological studies in one of the patients showed fibrotic lesions in the aortic valve cusps and in the aorta similar to those previously described by Bauer and his colleagues.

The Electrocardiogram in Myocardial Infarction.

M. M. WEISS AND M. M. WEISS, JUNIOR (*A.M.A. Arch. intern. Med.*, June, 1958) present a study of the electrocardiogram in myocardial infarction in a series of 304 cases in which the diagnosis was confirmed at autopsy. In every case abnormal electrocardiograms were recorded and changes considered to be specific indications of infarction were noted in 81·5% of these. In the remainder, the abnormalities were non-specific or were those of left bundle-branch block. The authors conclude that if serial records are obtained a normal electrocardiogram is never recorded in a case of myocardial infarction which is subsequently proved by pathological examination.

Carcinoma of the Pancreas.

D. BIRNBAUM AND J. KLEEBERG (*Ann. intern. Med.*, June, 1958) present an analysis of the clinical and laboratory findings in 84 cases of carcinoma of the pancreas from three hospitals in Israel. It was noted that this disease occurred mainly in Jews originating from European countries, and rarely in those from Oriental countries. Anorexia and loss of weight were the most frequent symptoms. The classical diagnostic syndrome of painless jaundice was rarely observed, but psychic symptoms occurred in 10 patients. Only about 50% of the patients with jaundice had a distended gall-bladder. Increased blood sedimentation rate was a very frequent finding. Hyperglycæmia or glucose tolerance tests giving a diabetic curve were observed in almost half of the patients, and were not correlated with the site of the lesion in the pancreas. External pancreatic insufficiency developing in the course of disease and proved by stool examination was found in 38% of the patients examined. Serum amylase determinations were found to be of limited value. Radiological examination pointed to the correct diagnosis in 25% of cases. The extremely high incidence of thrombo-embolic phenomena, especially in cases of carcinoma of the body and tail of the pancreas, is stressed as an important sign, and the role of trypsin in the thrombo-embolic process is discussed.

Allergic Reactions to Tranquillizing Drugs.

L. E. HOLLISTER (*Ann. intern. Med.*, July, 1958) discusses allergic reactions to tranquillizing drugs. Allergic reactions have been reported after the administration of three classes of these drugs which are widely used. These are (*a*) the phenothiazine derivatives (notably chlorpromazine), (*b*) the Rauwolfia alkaloids (notably reserpine), and (*c*) the substituted propanediols or butanediols (notably meprobamate). In the case of the first and third classes of drugs, allergic reactions account for the major complications encountered. Sensitivity to chlorpromazine

may be manifested by agranulocytosis, jaundice, dermatitis and various minor reactions. It is suggested that agranulocytosis from chlorpromazine is of immunological origin, though this remains to be proved, and that chlorpromazine jaundice is due to drug allergy. Reserpine is of low antigenicity, but occasionally angioneurotic œdema and urticaria have been reported. Two case reports are presented which indicate that purpura may be produced by the drug. Meprobamate sensitivity is manifested by fever, skin rashes, and constitutional signs including vascular collapse. This sensitivity is peculiar in that it is commonly produced by the initial dose of the drug. A case is reported that implies that the carbamate groups of the meprobamate molecule determine this type of sensitivity.

Diabetes Insipidus and Sheehan's Syndrome.

J. PIPER AND C. F. JØRGENSEN (*Acta med. scand.*, 1958, volume 42, fascicule 3) describe the occurrence of transient diabetes insipidus immediately after a post-partum hæmorrhage. Soon after this episode, which lasted three days, early signs of adenohypophyseal failure appeared; 10 months later the typical signs of Sheehan's syndrome appeared, and the diabetes insipidus had disappeared. The authors refer to histological changes in the neurohypophysis reported in patients who have died from Sheehan's syndrome. Presumably the diabetes insipidus disappeared because of adenohypophyseal failure. Cortisone therapy did not cause diabetes insipidus to reappear, probably because the secretion of anti-diabetic hormone was eventually taken over by the hypothalamus.

Hypercapnia.

W. T. THOMPSON *et alii* (*Amer. J. med. Sci.*, November, 1958) describe the effect of dichlorphenamide, a new and potent carbonic anhydrase inhibitor, in the treatment of patients severely ill with carbon dioxide retention complicating pulmonary emphysema and bronchitis. During ten days' administration of the drug there was a fall in the arterial carbon dioxide content and a rise in the oxygen saturation of the plasma, and there was much subjective improvement, with disappearance of wheezing. The authors are unable to state the mode of action of the drug. Its diuretic effect could not be wholly responsible (by drying out the lungs), for several patients who responded well lost very little weight. One of the drug's specific effects is to reduce bronchial spasm and secretion, and to facilitate alveolo-capillary diffusion of oxygen and carbon dioxide.

The Antecedents of Cirrhosis of the Liver.

L. OLHAGEN AND B. OLHAGEN (*Acta med. scand.*, November 10, 1958), investigating the previous medical history of 75 persons with cirrhosis of the liver who had never over-indulged in alcohol, or suffered from biliary disease, or from any other recognized antecedent of cirrhosis, found that they fell into several groups. In 12 patients severe or protracted bacterial infection had preceded the signs of liver disease, in 20 rheumatic disease (which in 12 cases was rheumatic fever) had done so, in four there had been renal disease, and in 13 there had been severe gastro-intestinal symptoms. Eight other patients had suffered from a variety of serious diseases, and in the remaining 13 patients the cirrhosis was regarded as cryptogenic.

Thyrotoxic Myopathy in the Elderly.

H. BOSTRÖM AND R. HED (*Acta med. scand.*, 1958, volume 42, fascicule 3) point out that myopathy, even when well developed, is easily overlooked in elderly patients, the changes seen being attributed to old age. This is especially true of thyrotoxic myopathy because thyrotoxicosis in the elderly may present few symptoms. Muscle biopsy shows only minor histological changes and clinical relief of the myopathic symptoms follows adequate antithyroid drug therapy.

Thrombosis and Gangrene Complicating "Q" Fever.

P. MICHON *et alii* (*Presse méd.*, December 25, 1958), in a report from the Pasteur Institute, describe a fatal case of "Q" fever with unusual features. The patient was a woman, aged 39 years, a farmer, who contracted an acute febrile illness of influenzal type. On the fourth day aphthous stomatitis made its appearance. Later, approximately during the seventh week, after a number of episodes of fever and stomatitis, hæmorrhagic patches developed and rapidly extended on the limbs; these were accompanied by blisters. At the same time, ulcers were noted in the mouth and on the genitals. The lesions rapidly assumed the appearance of necrotic purpura, particularly on the thighs. The patient's general condition deteriorated, and signs of myocarditis appeared. Skin grafts became necessary, as the lesions closely resembled severe and extensive burns. The patient was unable to tolerate the operative procedure and suddenly developed hypothermia and died. An autopsy confirmed the presence of myocarditis with enlargement of the liver, and revealed extensive thrombosis of the inferior vena cava. After other possible causes of the lesions had been excluded, the diagnosis of "Q" fever was proved by positive results of agglutination tests in the eighth and eleventh weeks at titres of 1/20 and 1/80 respectively. The patient was known to have been bitten by ticks on several occasions. Skin biopsy established the presence of thrombo-angiitis in the dermis and the epidermis. This accords well with the known damaging effect of the rickettsias on blood vessels. The authors state that peripheral lesions of the type observed in this case have not previously been described in cases of "Q" fever.

Diabetic Neuropathy.

M. ELLENBERG (*Amer. J. med. Sci.*, October, 1958) states that it has been generally assumed that the development of diabetic neuropathy is the result of diabetes poorly controlled over a long period. However, cases have been reported from time to time in which neuropathy was the initial clinical manifestation of diabetes. In the present paper he describes four cases in which the onset or exacerbation of diabetic neuropathy followed the institution of control of hyperglycæmia and glycosuria by insulin and diet. Insulin has been suspected as being the toxic ætiological agent under these circumstances, but the author points out that several factors seem to indicate clearly that this is not so. These are: (i) the neuropathy which follows the institution of insulin control is identical with all other forms of diabetic neuropathy; (ii) the neurological symptoms tend to diminish and even disappear despite the continued use of insulin; (iii) control of diabetes by measures other than insulin may also precipitate neuropathy. The author states that the ætiology of diabetic neuropathy remains obscure, but suggests that in the group of cases described a possible explanation may be in the sudden homœostasis, which produces the equivalent of a stress phenomenon. Support is given to this suggestion by the fact that stress situations such as surgery, infection and trauma, have been known to precede immediately the onset of diabetic neuropathy.

Serum Transaminase in Myocardia Infarction.

D. N. BARON *et alii* (*Quart. J. Med.*, October, 1958) discuss the value of serum transaminase estimations in the investigation of myocardial infarction. They find that in unequivocal myocardial infarction the serum transaminase level is always raised 12 to 48 hours later, and recommend serum transaminase estimation for the investigation of clinically doubtful cases of myocardial infarction, and consider that it may also have some prognostic value.

Spinal-Tap Headache.

R. J. BROCHER (*J.A.M.A.*, September 20, 1958) describes a technique to avoid spinal-tap headache. A small needle (number 18) should be used. After the needle is removed, the patient should lie on his abdomen for three hours. Among 894 patients, with the use of this technique, headache occurred only in four. In another group of 200 patients the supine position was adopted for three hours after the puncture, and among these patients 36% developed headache. The author states that leakage of spinal fluid has been suggested as the cause of headache in spinal tap. It was thought that lying on the abdomen after the tap would prevent leakage of spinal fluid and thus prevent headache. The author states that whatever the explanation, the results were convincing.

Mercurial Diuretics.

C. THORPE RAY (*A.M.A. Arch. intern. Med.*, December, 1958) discusses the mercurial diuretics, their mechanism of action, and their application. In spite of many years of clinical usage, there are several aspects of the mechanism of action of the mercurial diuretics which are unknown. The precise manner in which the inhibition of tubular reabsorption of sodium is effected, and the site of action on the renal tubule are not clearly established. However, the author concludes that these gaps in knowledge do not interfere with the clinical application of these very useful drugs.

On the Periphery.

BODY, MIND AND PATIENT: THOUGHTS PROVOKED BY A RECENT BOOK.[1]

A WORRIED-LOOKING man consulted his doctor. "Doctor, does worry cause indigestion, or is indigestion making me worried? Is my mind affecting my body, or the other way around?"

Now this was a serious question and deserved a serious answer. Fortunately the doctor was a serious man, who knew a little about philosophy, perhaps from his humanities course in medical school, and he replied: "I don't know whether I can answer that question. I mean, I'm sorry to say it's not a valid question. When you put up mind and body as alternative sources of your indigestion, I think you are making a fundamental 'category-mistake'. Mind and body can't be thought of as alternatives; they are in different categories." The doctor cast around for illustrations. "It's as if you asked me: which is more important to the cricket team, the team spirit or the slow bowling? Or, is the traffic noisier on bitumenized roads than in Sydney? These things are related, but they're in different classes and not comparable. That's why your question isn't valid."

"It's all right to ask you about my body?" queried the worried-looking man.

"Naturally. That's a valid question. You and I both know a lot about it. Your body's in space, in three dimensions. It's open to inspection—with your permission. It goes where your mind directs it. And as a doctor, I have a few extra bits of information about it—about your indigestion, for example."

"And it's all right to ask you about my mind?" faltered the worried-looking man.

"Of course—though there you have more information than I have. I can't experience your mind. You know your own thoughts, memories and feelings, perhaps not perfectly, but a lot better than I do. I know only what you tell me, or what I can pick up from outward signs, such as your behaviour."

"But I mustn't ask you about my body and my mind in the same breath?"

"Well, no—unless you want to fall into that category-mistake I explained to you."

"Oh, I don't want to do that. Thank you very much, doctor. I'll watch out for it. Goodbye."

The next doctor with whom the worried-looking man pursued the question was more at home in neurophysiology, cybernetics and the like than in philosophy. He countered: "It depends what you understand by your mind."

"What do you understand by a person's mind?" asked the worried-looking man, respectfully.

"Your mind is simply a set of functions performed by a particular organ: your brain. You have a digestive system, served by your gut and a few appendages, a respiratory system, by your lungs, and a system for information and communication, served by your brain. That's what you refer to as your 'mind'. It receives signs and signals, combines them with the information already at hand, and then may send a signal to the appropriate quarter, for action or otherwise. Not very mystical, really, though marvellously intricate in operation, as you can imagine."

"Marvellously", said the patient with indigestion.

The next doctor our persistent patient consulted, still hopeful, was a psychiatrist, to whom he posed a variant of the same question.

"Doctor, if a patient came to you very worried, run down and depressed, would you treat his mind or his body?"

The psychiatrist switched unobtrusively into the first person plural. "We could do either. We could suggest electric shocks, or tranquillizing pills—in which case we would be treating your body, obviously, or we could try talking about your experiences, your anxieties and your attitudes, trying to sort things out in your mind."

"Either treatment would make me feel better?"

"Well, yes—that would be the object of therapy. If successful, either the mental or physical treatment of your condition would make you feel better."

"Then is my condition mental or physical?"

"We don't use these words mental and physical much—we try to do away with them and, if necessary, use a word which includes aspects of both—'ego' for example. But if you want to understand how mental treatment or psychotherapy can affect your body as well as your mind, you might look at it this way: psychotherapy boils down to light and sound waves. Any new information you receive is carried by these waves, and that's physical enough. But that's pedantic; you're quite justified in regarding physical and mental treatment as something separate. Now, tell me, why did you ask?"

Our patient's search will obviously carry him further, but there is no point in following him on what may turn out to be for him a wild goose chase. He is imbued with the conviction of duality of mind and body, the "double-life" theory common to our culture, so that any number of painstaking doctors may not teach him a unitary concept of bodymind or mindbody. There may be no reason why, in the face of certain failure, they should try.

It may be expedient to respect his fanciful notion that he has a double life: that he has a body, which is in space and is like a machine, and that he has a mind, which is not in space, cannot be seen and is more like a ghost than a machine. It may even be appropriate for the doctor to share in the dogma of "The Ghost in the Machine", if that is the basic assumption on which a patient communicates ideas about health and sickness. Perhaps the doctor will even believe it himself—except in those austere official moments when he sighs for a Descartes to produce a new Authorized Version of the body-mind dilemma for up-to-date patients. And the soul—but we propose to leave theology out of it; if the worried-looking man should specifically bring it up on his next visit, we shall refer him to a specialist.

Adelaide. J. E. CAWTE.

[1] "Body and Mind in Western Thought: An Introduction to Some Origins of Modern Psychology", by Joan Wynn Reeves; 1958. Mitcham, Victoria: Penguin Books. 7" x 4¼", pp. 416, with illustrations. Price: 7s. 6d.

Medical Matters in Parliament.

HOUSE OF REPRESENTATIVES.

THE following extracts from *Hansard* relate to the proceedings of the House of Representatives.

March 18, 1959.

Model Health Acts and Regulations.

MR. WHITLAM asked the Minister for Health, upon notice:

1. When did the National Health and Medical Research Council approve model acts and regulations on radioactive substances, therapeutic substances, narcotic drugs and registration of births and fœtal deaths?

2. When were the model acts and regulations transmitted to the State governments?

3. Which States have enacted the model acts, and when did the State acts come into operation?

4. Which States have adopted the model regulations, and when did the State regulations come into operation?

DR. DONALD CAMERON.—The answers to the honourable member's questions are as follows:

1. The National Health and Medical Research Council recommended model acts in relation to radioactive substances on 19th May, 1954, and for narcotic drugs on 19th November, 1952. Model regulations for these two subjects were approved on 7th November, 1957, and 19th November, 1952, respectively.

2. Model acts on radioactive substances and narcotic drugs were transmitted to the States on 2nd July, 1954, and 4th November, 1952, respectively.

3. Amendments to give effect to the model act on radioactive substances were effective in Tasmania on 3rd September, 1956, Queensland on 1st July, 1958, and South Australia on 1st November, 1956. The necessary amendments have not become operative in New South Wales or Western Australia. No State has given effect to the model act relating to narcotic drugs.

4. No State has given effect to the model regulations relating to radioactive substances and narcotic drugs.

1-4. The National Health and Medical Research Council has not recommended model acts or regulations relating to therapeutic substances and registration of births and fœtal deaths.

Virus Influenza.

MR. UREN.—Would the Minister for Health inform the House what steps have been taken by the Department of Health to meet a possible outbreak of virus influenza, which has recently been raging in the United Kingdom? Have we sufficient vaccine available in Australia for inoculations, should the necessity for them arise.

DR. DONALD CAMERON.—Much attention has been given to this question, and quite large stocks of vaccine are held in the Commonwealth Serum Laboratories in Melbourne.

March 19, 1959.

Cortisone.

MR. BIRD.—I direct a question to the Minister for Health. Is the Minister aware that there is widespread concern among chronic asthma sufferers who are compelled to use cortisone to alleviate this distressing illness, at the high cost of this vital drug? Will the Minister take the necessary steps to secure the inclusion of this life-saving drug in the list of drugs provided free by the Government?

DR. DONALD CAMERON.—Cortisone and its derivatives are available as pharmaceutical benefits for treatment of the condition known as status asthmaticus. As the honourable member knows, it is not open to the Minister for Health to make additions to the list of pharmaceutical benefits unless such additions are recommended by the Pharmaceutical Benefits Advisory Committee, which consists of people in close touch with medicines and the practice of medicine. So far, the committee has recommended only that cortisone and its derivatives shou'd be made availab'e for treatment of this particular form of asthma, and until those gentlemen who are, I suggest to the honorable member, especially well-qualified to make the necessary judgement, recommend the inclusion of the drug for use in cases additional to those now covered, we have no power to include it in the list.

April 9, 1959.

Taxation.

MR. BROWNE.—As a large proportion of the cost of specialist medical treatment for people of the outback is incurred in travelling to a city for such treatment, will the Treasurer consider allowing such fares as a rebatable item for taxation purposes, when a patient is under doctor's orders to undergo specialist treatment?

MR. HAROLD HOLT.—The question on the face of it clearly indicates that it calls for some examination, and perhaps some rather technical examination. I do not quite know where such a proposal would lead. However, I shall see that it is examined, and that it is considered amongst other matters of policy which are normally considered at the time of preparation of the Budget.

SENATE.

THE following extract from *Hansard* relates to the proceedings in the Senate.

March 18, 1959.

Budgerigars and Poliomyelitis.

SENATOR TANGNEY asked the Minister representing the Minister for Health, upon notice.—

1. Has the Minister seen the report in the Melbourne "Herald" of 10th March of the possibility of a link between polio infection and budgerigars, such information being based on surveys made by doctors in Glasgow and Belfast?

2. In view of the great popularity of "budgies" as pets amongst those age groups most susceptible to polio, will the Minister investigate the position in Australia and ensure that the necessary steps are taken to minimize any danger that may exist.

SENATOR HENTY.—The Minister for Health has now furnished the following replies:

1. Yes.

2. The reports suggest that budgerigars may be susceptible to infection with poliomyelitis virus by members of an infected household. The role of the budgerigar in transmitting poliomyelitis is at the moment purely conjecture. However, a proved safeguard against infection with poliomyelitis is provided by Salk vaccine and unvaccinated persons of any age group will be at greater risk from human carriers than from budgerigars. In common with other parrots, the budgerigar is apt to be a carrier of psittacosis and bird fanciers kissing and fondling these pets thereby incur a greater risk from psittacosis than from poliomyelitis.

April 7, 1959.

Geriatrics.

SENATOR WOOD (through Senator Dame Annabelle Rankin) asked the Minister representing the Minister for Health upon notice.—

1. Has the Minister seen the claims made in the Sydney "Sunday Telegraph" of 1st and 8th March, relative to the many so-called miracle cures of the diseases of old age achieved at the Institute of Geriatrics, Bucharest, Rumania, by the use of the wonder drug H3?

2. Will the Minister have inquiries made into the authenticity of the claims and give consideration to the sending of a panel of health experts to the Bucharest Institute of Geriatrics to investigate the matter, and, if the reports are correct, to disseminate their findings throughout Australia?

SENATOR HENTY.—The Minister for Health has now supplied the following answers:

1. Yes.

2. The British Ministry of Health has studied reports of treatment carried out on the Continent with the use of the preparation known as H3. Expert opinion does not support the claims made for the value of treatment with this substance.

The studies made by the British Ministry of Health make it unnecessary for Australia to send a panel of experts to Europe, but if the claims for the drug are substantiated in the future, there is no doubt that it will become available in Australia.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE INCREASE OF TYPHOID FEVER IN MELBOURNE.[1]

[From the *Australasian Medical Gazette*, March, 1894.]

WE fancied that our southern sister had got over her troubles once and for all, as far as typhoid at least was concerned. The health reports for many months past have been satisfactory, and now, without any warning, the number of cases has enormously increased on a sudden, and the death rate for the month of January has gone up with a bound. Since the establishment of the new Board of Health, and the appointment of Dr. D. A. Gresswell to the position of Medical Inspector, a continuous crusade has been carried on against typhoid in Melbourne, and the measures taken to limit its ravages and to reduce the mortality have been admirable, and reflect the greatest credit upon the authorities engaged in the matter. It seemed almost a forlorn hope to purify a city like Melbourne, conceived in filth and disease, without drainage, except surface, and with a nightsoil service the most pestilential that was ever allowed to exist upon earth. But the Board of Health entered on its duties *con amore* nevertheless. It pointed out time after time that milk and drinking water must be boiled and filtered: that garbage must be destroyed by fire: that nightsoil must be removed in air-tight pans by daytime, and clean pans substituted for those removed: that some form of dry disinfectant must be used in privies to absorb noxious gases and smells: and chiefest of all, it took steps to have the fire-plugs removed out of the filthy water channels, in which they have been set in order to collect as much organic liquified matter as possible. The necessity for the latter procedure was shown in a most brilliant, and withal scientific manner, by M de Bavay, a pupil of M Pasteur, who was specially brought to Victoria for a brewery in Melbourne.

In the course of some bacteriological experiments, which he was conducting in order to establish the freedom from

[1] From the original in the Mitchell Library, Sydney.

various germs of the Yan Yean water before using it for brewing purposes, M de Bavay, to his amazement, encountered several pure cultures of typhoid bacilli in some water obtained on the spot. He repeated his experiments with success, showed his cultures to several gentlemen acquainted with the subject, and then made his discovery public. The tide of public indignation was strong against the scientist. The Government, at the recommendation of Professor Allen, sent to Sydney for a gentleman to disprove M de Bavay's assertions at a salary of £100 per month, and Dr. Katz and Professor Allen worked away at the problem with all their might. Notwithstanding that M de Bavay had to himself supply the pure cultures wherewith to start the control party, who evidently could not get them themselves in the then existing circumstances, the report found that M de Bavay's case was "not proven". And now the whirligig of time has at last brought that gentleman his revenge, for Dr. Gresswell only recently reiterated his warning that the water supply was the main source of typhoid contamination: that people would persist in drinking human excrement in spite of all warnings. Therefore de Bavay's typhoid bacillus must still be disporting itself in the limpid waters of the Yan Yean, and be supplied to the inhabitants without extra charge.

Correspondence.

ACUTE LARYNGO-TRACHEO-BRONCHITIS.

SIR: Acute laryngo-tracheo-bronchitis continues to kill more infants and young children than does diphtheria; a case of death from the latter makes headlines, because there is a prophylaxis. I am writing this letter because I believe I have found a cure for the former condition if the treatment is given early enough. I give intramuscular "Avil" and intramuscular tetracycline. The "Avil" somehow seems to relieve the upper respiratory obstruction, the patients losing their cyanosis in about 10 to 15 minutes, letting them live long enough for the antibiotic to be successful. I have now treated three cases in this manner without having to send them to hospital. The dose of "Avil" is: 1 ml. up to one year; 1·5 ml. from one to three years; 2 ml. over three years.

Might I add that in simple croup this drug is on its own, but unfortunately the oral form does not appear to work as well.

I do not suggest that we should attempt to treat laryngo-tracheo-bronchitis at home; I have only done so because I have always been very interested in this condition. However, I do suggest that if all practitioners were to give intramuscular "Avil" and intramuscular tetracycline before sending these cases to hospital, the death rate from this killer may be dramatically reduced.

Yours, etc.,

IAN H. WOOD.

Gilbert Street,
Latrobe,
Tasmania.
April 24, 1959.

EXTENDED RADICAL SURGERY IN THE TREATMENT OF CARCINOMA OF THE BREAST.

SIR: Dr. Mitchell's paper (MED. J. AUST., April 18, 1959) advocating extended radical mastectomy for carcinoma of the breast, can be regarded as an introduction to a further manœuvre in the surgical treatment of some selected cases. It should be emphasized that it can be applicable to only a small proportion of cases, and is yet in the period of clinical trial. Dr. Mitchell admits that we will have to wait until 1962 before any assessment of its value can be produced. However, in the course of his paper, he has made several statements about the present methods of therapy, and suggests a plan of treatment for cancer of the breast in general which differs considerably from those in present common use. Some of these points merit further discussion.

Firstly, he states that after radical mastectomy, axillary recurrence is rarely seen. This is true up to a point. Stage II includes a range of nodal involvement from the barely palpable to the barely operable. In the former, it may be safe to leave the axilla unirradiated; in the latter, it must be rare for a surgeon to have such confidence in his dissection that he does not refer his patient for post-operative radiotherapy.

Secondly, it has become fashionable to attempt to discredit the work of Professor McWhirter by stating that other workers have not been able to reproduce his results. While not myself entirely convinced that his method is always ideal for every case, it should be made clear that those who fail to achieve good results may not, in fact, be using McWhirter's technique at all. For example, consider the simple mastectomy incision. For adequate radiotherapy, it must be vertical. Horizontal incisions, often extending to the posterior axillary line, make effective radiotherapy virtually impossible. Yet these and other bizarre incisions are repeatedly seen, in this city at least. In other centres "which have failed to reproduce McWhirter's results", it may equally be so: if this be true, then relatively poor results would not be surprising. Further, Professor McWhirter does claim sterilization of lymph nodes with X-ray therapy; otherwise the whole procedure would be futile. This admittedly hinges on what constitutes a "viable cell". An irradiated cell, seen in histological section, which was clearly "alive" at the time of excision, and yet has been so altered by ionizing radiation that it is no longer capable of growth, can hardly be called "viable" in the neoplastic sense.

Thirdly, the increased morbidity of extended radical mastectomy is justified on the ground that the alternative method of therapy (i.e., radiation) produces an equal morbidity with pulmonary fibrosis. While it is true that fibrosis of the apex and of the most superficial parts of the anterior lung, of sufficient degree to produce symptoms, is occasionally seen, it is by no means common. Dr. Mitchell's plan of treatment includes such X-ray therapy in addition to the extended operation; it is thus not an alternative, but an additional hazard. Further, when radical mastectomy has been done for a strictly operable tumour, and the only doubt lies in probable involvement of the internal mammary chain, then it is only necessary to irradiate these nodes. This involves the irradiation of relatively little lung tissue.

Fourthly, Dr. Mitchell states that 15% to 20% of patients develop recurrence on the chest wall. Another way of stating this would be that 15% to 20% of patients submitted to radical mastectomy were inoperable. A radical cancer operation which leaves tumour in the primary site can never be expected to improve the patient's chances of survival. To operate on these cases, and then to expect to avert the consequences by applying radiation to a surgically devitalized area, is to expect the impossible.

Finally, it is stated that the outcome of the treatment of cancer of the breast is dependent on the efficacy and radical nature of the first operation. This in itself is open to question; a great deal depends on the nature of the disease process itself, of which we are at present largely ignorant. Some recent papers suggest that the opposite may be true.

Despite these criticisms, it does appear that there may be a place for this manœuvre in the management of a proportion of cases of breast cancer; there can be none who would disagree that it is a method which should be fully investigated. It is to be hoped that this investigation will contain a control group of cases treated with radiation to the internal mammary chain. Lack of controls is, to my mind, the only really valid criticism which has been levelled at the work of Professor McWhirter.

Apart from this, the general plan for treatment set out in the paper contains many departures from conventional therapy. These need more critical assessment before being adopted by any surgeon or radiotherapist.

Yours, etc.,

D. P. EWING.

Royal Prince Alfred Hospital,
Camperdown, N.S.W.,
April 21, 1959.

THE STUDENT HEALTH SERVICE.

SIR: I would refer to your leading article of April 18, 1959, concerning the student health service to be established within the University of Sydney. You stated that the full-time appointment of a Director of Student Health was, in your belief, the first of its kind to be made in Australia.

The establishment of such a service is to be commended, but I should like to place on record the fact that a student health service for students attending teachers' colleges in

New South Wales was established in 1913. Prior to this, commencing in 1896, some lectures had been given to trainee teachers on school hygiene, and in 1904, Dr. Mary Booth was appointed as the first lecturer in school hygiene to the teachers' college, which was at that time situated at Hurstville. Students were medically examined as early as 1906. With the constitution of the School Medical Service in 1913, under the late Dr. C. Savill Willis, an organized system of health supervision of students was inaugurated, and has continued to the present time.

This service provides a medical staff to the various teachers' colleges to supervise the students' health, to counsel students seeking advice or referred by the teaching staff, and to lecture on health education.

The total number of students is approximately four and a half thousand, and eight medical officers—of whom three are part-time—are attached to the various colleges. All students are given a complete medical examination prior to entrance to college, and the medical record for each student is maintained throughout his course. The medical officer is available to carry out first-aid treatment, if necessary, but no other treatment is undertaken.

The service has proved an exceedingly successful one, and I feel sure that my medical officers will look forward to the arrival of Dr. Malleson, and will be glad to receive the benefit of his experience.

Yours, etc.,

E. S. A. MEYERS,
Director.

School Medical Service,
Department of Public Health,
86-88 George Street North,
Sydney.
April 23, 1959.

THE SURGERY OF PENO-SCROTAL HYPOSPADIAS.

SIR: While I do not wish to enter into a long-continued controversy on the problem of repair of hypospadias, I feel in justice to myself that I must crave space to reply to some of Mr. Denis Browne's remarks in his letter which appeared in THE MEDICAL JOURNAL OF AUSTRALIA of May 16, 1959.

In referring to my article "Hypospadias" in the *Australian and New Zealand Journal of Surgery*, 1958, Volume 27, Number 4, Mr. Browne has taken some of my words out of context, and, quite inadvertently I am sure, he appears to have done what he complained of MacCollum doing—that is, not reading the text carefully enough.

I did say that in his operation "The scrotal skin is dragged up on to the penis . . . This however does not result in any functional disability".

In the next paragraph, and when dealing with quite a different aspect of his technique, I went on to say: "The epithelium which has to grow round to line the roof of the new urethra is in juxtaposition to the ventral surface of the penis and any failure at all of perfect skin union on the ventral surface of the penis quickly results in the formation of a short epithelial lined track from the new urethra to the surface with the inevitability of a persisting fistula." It is obvious that here I was referring to penile skin, and not to scrotal skin as Mr. Browne implies in his letter.

I stress this point, because the failures surgeons experience in carrying out his operation are not so much in the build-up to the peno-scrotal junction, when such is necessary, but rather in the build-up from the peno-scrotal junction, or just in front of it, to the tip of the penis, and this is where he uses penile skin flaps united in the mid-line to cover the under-surface of the penis.

Turning now to his criticism of the photographs of the patients in my series, in which he states the "results show a very short undersurface and a surplus of unused preputial skin on the upper side", this is an understandable misstatement which I really must contradict.

Mr. Browne will be the first to agree that it is extremely difficult to convey an accurate impression of the actual shape of a small boy's penis when taking a photograph of him micturating in the presence of arc lights and an interested audience.

I can assure Mr. Browne—and this has been confirmed by many surgeons who have seen these cases—that my statement "The results functionally and cosmetically have been satisfactory" is an absolutely true statement in all its implications. Evidently this is the opinion also of Dr. W. W. Scott, Editor of the "Year Book of Urology", who has published the photographs in question in his 1958-1959 edition.

Since the completion in September, 1957, of the series dealt with in my paper, I have continued to use the same technique with no variation at all, and up to the present time there has been no fistula formation nor indeed a complication of any kind.

It is indeed interesting to read Mr. Browne's statement that Sir Archibald McIndoe, a preeminently skilful surgeon, has now abandoned the inlay graft which he employed for so long.

I am a great admirer of Mr. Denis Browne's achievements in many branches of pædiatric surgery, and am very impressed with the successful results he can achieve in hypospadias repair using his own method. It is my considered opinion, however, that in carrying out his particular operation very few, if any, surgeons can achieve results comparable with his own.

If there are two operative techniques under consideration and the first one gives consistently better results in the way of primary healing without fistula formation than does the second one, in the hands of what might be termed the average competent and experienced surgeon, then there seems no doubt in my mind that the first operation is obviously the proper one for general adoption, for the simple reason which I stressed in my paper—that a wide margin of safety is better than a small one where the avoidance of fistula formation is concerned.

For this reason I venture to predict, without in any way belittling Mr. Denis Browne's eminence as a surgeon and with due regard to the satisfactory results which he himself can attain, that in another thirty years his hypospadias operation will have gone the way of the inlay graft, and that one or other of the modifications of the Cecil operation, much as I described in it *The Australian and New Zealand Journal of Surgery*, (1958, Volume 27, Number 4), will have been more or less uniformly adopted as the method of choice for hypospadias repair from the peno-scrotal junction forwards.

Naturally Mr. Denis Browne will not agree with me about this; but perhaps he will at least be in accord with the principle that any surgeon is well advised to persevere with a technique which both anatomically and functionally and in the absence of complications, has proved eminently satisfactory in his own hands.

After all, there is still a lot to be said for the old Roman proverb: *"Ne sutor supra crepidam"*.

Yours, etc.,

KENNETH FRAZER.

Ballow Chambers,
Wickham Terrace,
Brisbane.
May 18, 1959.

EXTENDED RADICAL SURGERY IN THE TREATMENT OF CARCINOMA OF THE BREAST.

SIR: The address of Dr. R. I. Mitchell, F.R.C.S., F.R.A.C.S., on "Extended Radical Surgery in the Treatment of Carcinoma of the Breast", as published in THE MEDICAL JOURNAL OF AUSTRALIA, April 18, 1959, page 527, was interesting and thought-provoking.

The fact that I disagree with Dr. Mitchell that the Urban operation has a place in the present-day treatment of carcinoma of the breast is, I am sure, of little moment to him. The ultimate acceptance or rejection of this operation will, as Dr. Mitchell points out, only be decided after a sufficient interval for its results to be assessed. However, there are a few comments I would like to make in respect to this paper.

1. Surely pulmonary fibrosis after irradiation to the internal mammary nodes is no problem in this modern age, when the multi-slanting beam technique is used.

2. Efficient irradiation to the axilla post-operatively is, in my opinion, completely without danger, and there is no question that it increases the survival rate. It is practised on a wide scale and, to my knowledge, without harm.

3. Dr. Mitchell's claim that prophylactic castration in no way alters the behaviour of the disease does not concur with the experience of Patterson of Manchester.

4. My main objection to the Urban operation is that it seems on anatomical and clinical grounds not to be sufficiently radical. The internal mammary chain of lymph

nodes extends well above the second costal cartilage. One of the commonest sites for recurrent glands to become evident is behind the lower end of the sterno-mastoid. This corresponds with the upper limits of the internal mammary nodes, well above the area of the Urban excision. For this reason I am not confident that the extended radical mastectomy will ever establish its place in the treatment of carcinoma of the breast. I agree with Dr. Mitchell that there seems to be no place for the Wangensteen operation, because of its magnitude.

5. I feel that Dr. Haagensen's attitude is the correct one. If frozen section reveals involvement of the internal mammary nodes, I think experience will prove the case to be incurable by surgery. Dr. Haagensen's method of biopsy of the internal mammary nodes is surely similar to that originally used by R. S. Handley.

Yours, etc.,

JOHN CONNELL.

70 Collins Street,
Melbourne,
April 28, 1959.

GROWING UP IN A CHANGING WORLD.

SIR: I have just read with interest your admirable account of the tenth annual meeting of the World Federation for Mental Health under the title of "Growing up in a Changing World" (March 7, 1959). That section of your article that reports my answer to a question as to the value of corporal punishment in institutions for the treatment of juvenile delinquents is an accurate condensation; but, in its condensed form and shorn of one or two touches of humour and outside the context of a panel discussion in an international conference, it leaves a more forceful impression than I would care to have to justify in general. I would like to emphasize, if I may, that my forceful opposition to corporal punishment is restricted to corrective institutions for juvenile delinquency, and I certainly stand by my opinion that where there is no relationship of love and trust between the punisher and the punished, that corporal punishment is apt to have a brutalizing and perhaps corrupting effect on both parties, which I believe far outweighs any possible good results.

It is true, also, that in general I am against the use of corporal punishment as not leading in the direction in which most of us would like our civilization to advance; but I would hesitate to commit myself to being "definitely, totally and absolutely against it" in all circumstances.

Yours, etc.,

KENNETH SODDY, M.D.,
Scientific Director.

World Federation for Mental Health,
19 Manchester Street,
London, W.1, England.
May 11, 1959.

CANCER, A DISEASE OF THE NERVOUS SYSTEM.

SIR: Chronic irritation long-continued, whether mechanical, chemical or bacterial, seems to be the only cause of cancer yet agreed to. Cancer can thus be produced experimentally. But how does this chronic irritation so act that it alters the metabolism of the cell from normal to malignant?

I submit that the process may be due to loss of trophic nervous control of the metabolism of the cell or cells (because, as recently suggested, malignant changes may begin not only in a single cell, but may appear simultaneously in a group of cells). Long-continued irritation of the trophic fibres of nerves may so upset their control as to allow excess of nutrient and oxygen to pass through the cell envelope to the nucleus, resulting in hyperactivity, growth and reproduction (this may simulate, in some degree, what happens to the fertilized ovum). Amongst the waste products of this raised cell metabolism and the increased intracellular oxygen, it may be that the CO_2 is returned to the circulation to be exhaled from the lungs; but there may be many monoxides returned to the circulation to form stable products, of a toxic nature, with the haemoglobin, and these gradually accumulating produce that cachexia, loss of tone and malaise so well known in the cancer patient.

This theory of loss of trophic nerve control of the cell explains much of the behaviour of malignancy.

But there are many questions I should like to ask the research worker with whom this conception must now be developed.

Yours, etc.,

LLEWELLIN DAVEY, M.B., B.S., F.R.C.O.G.

183 Payneham Road,
St. Peters, South Australia.
April 18, 1959.

Medical Practice.

THE PROMOTION OF ETHICAL DRUGS: CAUSE AND EFFECT.

ON the evening of Wednesday, March 25, in the Pharmacy School, University of Sydney, a symposium on "The Promotion of Ethical Drugs: Cause and Effect" was held under the auspices of the Institutional and Industrial Pharmacists' Group (incorporated in the Pharmaceutical Society of New South Wales) before an audience of members and visitors. Under the chairmanship of Mr. P. A. Smith, managing director of Burroughs Wellcome & Co., the following panel spoke in the order given: Dr. H. P. B. Harvey, physician; Dr. T. St. Leger-Moss, general practitioner; Mr. W. B. Howard, managing director of William R. Warner & Co. Pty. Ltd.; Mr. J. P. Fetherston, Ph.C., Royal North Shore Hospital of Sydney; Mr. L. Lewis, of Russell Lewis & Associates, Advertising Consultants. The views expressed by the speakers were entirely personal and did not reflect those of any official body.

The contributions of the panel and the discussion which followed clearly indicated several important points:

1. The medical profession, pharmacy and the pharmaceutical industry were each an integral part of the "health team" and, in that sense, dependent upon one another.

2. There was complete unanimity of desire to attain a maximum of efficient cooperation between all three, particularly in respect of the promotional activities of the industry, but not so complete an appreciation of each party's problems and requirements.

3. Drug firms should supply their product information in as concise a form as possible, consistent with completeness and accuracy. The specialist in particular asked for substantiating evidence for the claims made, not merely graphs or tables in eye-catching colours but with no legend accompanying them.

4. The role of advertising in the mass dissemination of product information to doctors and pharmacists was recognized. Both medical opinion in the symposium and evidence produced by a survey conducted in Great Britain by Vitamins Ltd.[1] indicate clearly that doctors and pharmacst have a preference for personal discussion with drug firm representatives. Mail matter and journal advertisements come second and third respectively.

5. The role of the pharmacist in this connexion was discussed at some length. It was suggested that he had a part to play in three spheres, as the drug firm representative, as the hospital pharmacist and as a retail pharmacist.

6. The pharmacist asked that, in addition to therapeutic information, the industry should provide him with pharmaceutical information about drugs and drug products.

The conclusions to be drawn from these points make a very strong case for more constructive thinking and action on this major problem of ensuring rapid, complete and accurate dissemination of information by the drug industry to those who alone can utilize it. They surely indicate a need for further cooperative action by all three bodies with a view to achieving some resolution of the present difficulties. More efficient and effective liaison, aimed at an objective improvement in therapeutics, must reflect to the benefit of the community and the parties concerned.

Public Health.

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL.

THE forty-sixth session of the National Health and Medical Research Council was held at Sydney on November 18, 1959. The Council confirmed the following resolutions:

Resolution 1: That Œstrogens, Progesterone and Androgens or their preparations should be sold only on prescription and only be repeated on prescription.

[1] *Brit. med. J.*, 1958, 2 (November 1).

Resolution 2: That the Commonwealth Department of Health make funds available annually to send an Australian representative to the United States to attend the Federal Drug Administration's International Conference on Antibiotics.

Resolution 3: That broad spectrum antibiotics should be made available free in quantities up to 100 capsules for the long term treatment of bronchiectasis and fibrocystic disease of the pancreas.

Resolution 4: That Item 37 of the Second Schedule should be amended to permit issue of 100 tablets of Penicillin V for the prophylaxis of nephritis and rheumatic fever.

Resolution 5: (*a*) All States adopt the list of Notifiable Diseases submitted in 1950 as amended from time to time by resolution of the National Health and Medical Research Council, (*b*) the law requiring notification should discriminate between—(i) diseases required to be notified for the information of the Central Health Authority, (ii) diseases required to be notified for purposes of statutory prophylactic measures by the local health authority or the Central Health Authority.

Resolution 6: (i) That all types of leukæmia should be made notifiable in every State, (ii) that the information to be collected about each notified case and the reason for requiring it should be set out in a document to be prepared for the Council by Sir Macfarlane Burnet, Dr. E. V. Keogh and Professor E. S. J. King, (iii) that each State should set up a consultative panel to collect and elucidate this information.

Resolution 7: That it re-affirm its recommendation for the early immunization of the age group 15-44 years of age against poliomyelitis.

Resolution 8: That States require notification of breast abscess and of acute infections in the new born occurring in the first four weeks after birth, and including mastitis, conjunctivitis, panonychia, dermatitis, infection of the cord and any systemic extension of these to pneumonia or enteritis.

Resolution 9: That the National Health and Medical Research Council re-affirm its recommendation of May, 1957, that States arrange for decentralization of supplies of Gamma Globulin. The Council further recommends that with the cooperation of the British Medical Association and THE MEDICAL JOURNAL OF AUSTRALIA, States undertake a campaign to achieve the wider use of Gamma Globulin in the prophylaxis of infectious hepatitis.

Notes and News.

The Medical Women's Society of New South Wales: Annual Prize.

The annual prize of the Medical Women's Society of New South Wales for 1958 has been awarded to Dr. Patricia Davey, Dr. Clair Chalmers and Dr. Minna Golomb for their work on "Acute Otitis Media in Children."

Standard for Babcock Testing of Milk.

Many commercial transactions in milk and milk products involve fat determinations by the Babcock method. The publication of an Australian standard specifying the glassware and methods for Babcock testing will therefore be of interest to producers and purchasers of milk products. The standard has been issued as A.S. No. N.26-1958, with the title "Glassware and Methods for the Determination of the Percentage of Fat in Milk, Skim Milk, Separated Milk, Butter Milk and Cream by the Babcock Method".

In the past, Babcock testing had been carried out substantially in accordance with the relevant British specification B.S. 755-1937. It has long been recognized that the results so obtained are consistently higher than those given by the gravimetric ether-extraction reference method. The preparation of the Australian standard was undertaken with the object of improving the accuracy and reproducibility of the test. The new Australian standard is based on B.S. 755, but differs from it in certain significant details. In particular, it provides that the level of the liquids in the pipette be read to the top of the meniscus both in the calibrating of the pipette and in the measuring out of the test sample, and that the sample be pipetted at a temperature within the range 38°-40° C. (100°-104° F.). It also includes more detail regarding the methods of reading the fat column than is given in B.S. 755.

Regulations in each State require government certification before the sale of glassware to be used for Babcock testing in connexion with commercial transactions. It is hoped that this standard may provide a common basis for such certification. Copies of A.S. No. N.26 may be purchased from the headquarters of the Association, 157 Gloucester Street, Sydney, and from branch offices in capital cities and at Newcastle. The price is 7s. 6d.

The Orange Cross: International Jubilee Conference.

An international conference to celebrate the jubilee of the Royal National Society for Life-Saving and First Aid (The Orange Cross) of the Netherlands will be held from September 10 to 12, 1959, at Scheveningen, The Hague, under the patronage of Her Majesty Queen Juliana of the Netherlands. A particular object in the organizing of this conference is to help to restore the international contact between life-saving societies which was broken by the Second World War.

The main subjects for discussion will be resuscitation, burns and electric shock. Seven "case histories" will be presented to illustrate new or special developments in the field of first aid in accidents. There will also be international first aid competitions, and a formal meeting to reestablish the "International Association of Life-Saving and First Aid in Accidents".

Further details of the conference may be obtained on application to the Secretary, H. J. Oosterhuis, at the following address: 14, Burgemeester de Monchyplein, The Hague, Netherlands.

An Australian Orthopædic Association Prize.

The Australian Orthopædic Association Prize, the sum of 50 guineas, will be awarded every two years for an original and unpublished essay on some orthopædic subject, to be either nominated by the Committee of the Association or left to the choice of the candidate. On this occasion the choice of subject is left to the candidate. Entrants for the prize are to be either sixth-year medical students or graduates of not more than five years' standing. The essay shall not exceed 5000 words. If no essay is deemed of sufficient merit, the committee retains the right to withhold the prize. The publication rights of the essay shall belong to the Australian Orthopædic Association. Essays should be typewritten with double spacing, and should be in triplicate; they should be forwarded under a pseudonym to the Honorary Secretary of the Australian Orthopædic Association, Dr. Richard Hodgkinson, 135 Macquarie Street, Sydney. A separate sealed envelope should be enclosed, containing the name and address of the candidate together with the pseudonym used. Entries for the prize will close on June 30, 1960.

A New Medical Centre for Macquarie Street.

The foundation stone of a modern 12-storey medical building, to be called the William Bland Centre, was laid on May 22, 1959, by Sir Charles Bickerton Blackburn, Chancellor of the University of Sydney. Situated opposite Sydney Hospital and the Mint Building, the new building has been designed by the architects, Messrs. H. P. Oser and Associates, to provide members of the medical and allied professions with unit-type accommodation based on the home unit principle. It will fit in with the conservative atmosphere of Macquarie Street and yet at the same time present a modern appearance with attractive clean-cut lines. Construction will be by the lift-slab method. The front will consist of aluminium curtain walling with ceramic armourcast spandrel panels. Heat-absorbing glass has been used in the windows, which are so designed that individual air-conditioning units can be installed without alteration. The sides and rear of the building will be face brick with metal windows.

The building will consist of 12 floors including basement and ground floor. The upper ten floors of the building will contain 140 units, which range from 271 square feet for single suites to 794 square feet for triple suites. Each floor will contain four single, four double and two triple suites. Each suite will contain a waiting room, examination room and consulting room, and will be situated so as to give the maximum of natural light.

The ground floor and basement will be used for commercial purposes allied to the medical profession and in keeping with the general prestige of the building. The Bank of N.S.W. will have a branch here. Two high-speed automatic

lifts will be provided, one being capable of accommodating an ambulance stretcher or 20 passengers, and the other of accommodating 13 passengers and a lift driver. A cleaning room on every floor will have access to an incinerator chute leading to an incinerator installed in the basement.

The Centre was named in honour of Dr. William Bland, a naval surgeon who was transported to Sydney from England in 1814 after killing a man in a duel. He was given a pardon a year after his arrival and became the colony's first full-time medical practitioner.

Fifth International Convention on Nutrition and Vital Substances.

The International Society for Research on Nutrition and Vital Substances, which was formed four years ago, will hold the fifth International Convention at Constance (Germany) and Zurich (Switzerland) from October 7 to 11, 1959. The suggested subjects for discussion are as follows: the soil of the life cycle (plants, animals, man); the doctor and the nutritional and developmental problems of adolescence; water; radiations (physical, biological and medical aspects); disturbances of radiation potential due to atom-splitting; milk, bread, grain; the whole question of toxicity, a cause of the diseases of civilization; cancer, a universal disease; the teeth, a factor in biological wholeness; caries, primarily conditioned by civilization; nutritional reform. The following are members of the committee: Professor H. A. Schweigart (Hannover, Pretoria), Professor W. Heupke (Frankfurt), Professor Cl. Antoniani (Milan), Professor D. G. Steyn (Pretoria), Dr. H. Warning (Frankfurt), Dr. S. Klein (Brussels). Further information about the convention may be obtained on application to the following address: The Secretariat, Internationale Gesellschaft für Nährungs und Vitalstoff-Forschung E.V., Hannover-Kirchrode, Bemeroder Strasse 61, Germany.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 17 of March 5, 1959.

NAVAL FORCES OF THE COMMONWEALTH.

Appointments.— David Alexander Noble is appointed Surgeon Lieutenant (for Short Service) (on probation), dated 5th January, 1959.

Termination of Appointment.—The appointment of George Graham Mahony, as Surgeon Lieutenant (for Short Service), is terminated, dated 26th December, 1958.

Citizen Naval Force of the Commonwealth.

Royal Australian Naval Reserve.

Promotions.—Surgeon Lieutenant William George Tellesson is promoted to the rank of Surgeon Lieutenant-Commander, dated 21st December, 1958.

Royal Australian Naval Volunteer Reserve.

Termination of Appointments.—The appointments of the following are terminated, to date 31st March, 1958:—Surgeon Lieutenant-Commanders Stewart Horton Delbridge Preston, Norman Alfred Richards and Peter Donald Graeme Fox.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 21, of March 25, 1959.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

R.A.N.R.

Appointments.—Surgeon Commander Graeme Lindsay Grove, Royal Australian Naval Volunteer Reserve, is appointed Surgeon Commander, with seniority in rank of 30th June, 1955, dated 19th January, 1959.

District Naval Medical Officer.—The appointment of Surgeon Lieutenant Andrew Keith Frazer, as District Naval Medical Officer, Western Australia, is terminated, dated 11th January, 1959.

Resignation.—The resignation of Donald Metcalf of his appointment as Surgeon Lieutenant is accepted, dated 12th January, 1959.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Ronald John Smith (0310774) is appointed to a short service commission, on probation for a period of twelve months, 7th October, 1958, with the rank of Flight Lieutenant.

The short-service commission of Flight Lieutenant A. M. Muirhead (042575) is extended to 14th August, 1962.

Active Citizen Air Force.

Medical Branch.

No. 21 (City of Melbourne) Squadron.—Flight Lieutenant I. G. Nicol (0312763) is granted the acting rank of Squadron Leader, 14th October, 1958.

Air Force Reserve.

Medical Branch.

George Edward Straffon (257993) is appointed to a commission, with the rank of Flight Lieutenant, 14th October, 1958.

Harold Ludivic Rowe Story (257992) is appointed to a commission, with the rank of Flight Lieutenant, 26th September, 1958.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-end Course in Gastro-Enterology.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in gastro-enterology for general practitioners will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, Camperdown, under the supervision of Dr. Stanley Goulston, on Saturday and Sunday, July 4 and 5, 1959. The programme will be as follows:

Saturday, July 4, chairman, Sir William Morrow: 9.30 a.m., "Recurrent Abdominal Pain in Childhood", Dr. Wilfred Cary; 10.15 a.m., "Hæmatemesis and Melæna in Childhood", Dr. Eric Goulston; 11.30 a.m., "Too Much and Too Little Acid", Dr. Douglas Piper; 12.15 p.m., "Constitutional Factors in Peptic Ulceration", Dr. Brian Billington.

Sunday, July 5, chairman, Sir William Morrow: "Advances in Techniques in Routine Hospital Investigations": 9.15 a.m., (a) "Radiology", Dr. A. R. Colwell; 9.45 a.m., (b) "Laboratory Methods", Dr. Mark Playoust; 10.15 a.m., (c) "Biopsy Studies", Dr. J. Rankin; 10.30 a.m., discussion. "Advances in Therapy": 11.30 a.m., "The Alimentary Canal", Dr. Bruce Hall; 12.15 p.m., "The Liver and Pancreas", Dr. Richard Boden; 2 p.m., "Pitfalls in the Surgery of Gall Bladder Disorders", Sir William Morrow; 2.45 p.m., "Why Surgery May Fail to Cure a Duodenal Ulcer", Professor John Loewenthal; 4 p.m., "How Should One Manage a Case of Obstructive Jaundice", Dr. Stanley Goulston and Dr. Norman Wyndham.

The fee for attendance is £3 3s., and those wishing to attend are requested to make written application, enclosing remittance, to the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic Address: "Postgrad Sydney".

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales, in accordance with the provisions of the *Medical Practitioners Act*, 1938-58.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1a) of the Act: Antonenko, Nikolai Pavlovich, M.B., B.S., 1959 (Univ. Sydney); Faine, Solomon, M.B., Ch.B., 1950 (Univ. New Zealand); Pozzi, Phillip Anthony Michael, M.B., B.S., 1954 (Univ. Queensland); Yaxley, Ronald Peter, M.B., B.S., 1950 (Univ. Queensland), F.R.C.S., Edinburgh, 1955, F.R.C.S., England, 1956.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1b) of the Act: Davies, Ian Douglas, M.R.C.S., England, L.R.C.P., London, 1953, D.M.R.D. (R.C.P. & S.), 1958; Goldacre, James Alfred Henry, M.R.C.S., England, L.R.C.P., London, 1948; Lyne, Alan John, M.B., Ch.B., 1956 (Univ. Birmingham); Wade, Roger Noel, L.M.S.S.A., London, 1954, M.B., B.Ch., 1955, B.Sc., 1951 (Univ. Wales); Tan, Dixie, M.B., B.S., 1958 (Univ. Malaya).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (2A) of the Act: Hatos, George, M.D., 1937 (Univ. Szeged); Hollo, Stephen Julian, M.D., 1950 (Univ. Budapest); Szekulesz, Istvan, M.D., 1950 (Univ. Budapest).

Registered medical practitioners who are required to comply with the requirements of Section 17 (3) and are registered under Section 17 (1a), (the qualifications being M.B., B.S., 1959 (Univ. Sydney)): Adler, Edith Marion; Ahearn, Raymond Stephen; Allardyce, Clive Frazer; Amos, Bernard John; Appel, Denis Anthony; Barnes, Peter Henry Markham; Baume, Peter Erne; Baumgarten, Alexander; Benjamin, David; Best, Janice Ann; Biddulph, Robert Arthur John; Bradhurst, Peter Graham; Burgess, John Austin; Burgheim, Eva; Burns, Michael Warren; Cameron, Basil Geoffrey; Carlton, Mark Anthony; Childs, Winifred; Chong, John Yok Hong; Chung, Eng Chieuh; Cocking, Keith Josiah; Conolly, William Bruce; Cooke, Allan Roy; Cossart, Yvonne Edna; Coupland, Graham Arthur Edwin; Coupland, William Warwick; Crago, Richard Chilton; Cronan, John Patrick; Crowley, Paul Bertram; Dalton, Vincent Cletus; Dight, Ronald; Dowsett, John; Duffy, Gavan William; Erby, Angela Jane; Evans, Richard Anwyl; Faithfull, Donald Kingsley; Faunce, Edmund Alured de Laune; Field, Geoffrey Burstal; Fink, Norbert Frederick Raoul; Finlayson, Paul Farrington; Fixler, Sylvia; Flynn, Gerald James Livingstone; Ford, Selina Joan; Freeman, Edward Alan; Gallagher, Pamela; Garlick, David George; Gerber, Nicholas; Gibson, James Sedman; Gilder, Esther Mary; Gluskie, Clarence Alexander; Godfrey, Howard Fuller; Gong, Laurence William; Goulston, Kerry John; Green, Stanley; Harrison, Owen Michael; Haskins, Jan Margaret; Hennessy, Brian Leslie; Herriott, Bruce Arthur; Hobbes, Alick Frederick Truscott; Hooper, Nanette Catherine Mary; Houghton, Graeme Warwick; Hunt, John Hamilton; Hyde, Trevor Francis; Jacob, Kurishingal; James, Owen Francis; Johnson, Lindsay Albert; Jones, Donald George; Joseph, Cappil Philip; Kahans, Daniel; Kuo, Dick; Kwok, Frank Yam Wang; Lam, Thomas Sit-Tin; Landecker, Kathrine Dorothy; Lee, Ang Kim; Lee, Celia Margaret; Lee Kheng-Chew; Little, John Miles; Long, Geoffrey Joseph; Lovric, Ivan; Ma, King Yuk; McCarthy, William Henry; McCredie, Janet; McDonald, Robert Concord; McGrath, Philip James; McGree, Maurice Dening; McGuire, Peter Neville; McInerney, Valerie Jean; McKee, John Douglas; Macken, James Ernest; McKeown, Diarmid Ronald; McKinnon, Malcolm Graeme; McKinnon, Ronald Marius; McLachlan, John; McLeod, James Graham; McNamee, John Thomas; Madew, David; Nanoliaras, Peter Theodore; Marek, Zdzislaw; Mitrofanis, Christos; Moran, Barry Edward Francis; Moran, Clement Charles; Morony, Margaret Leone; Morris, John George; Morris, Peter Leo; Moulton, William Kirton; Mowbray, Graham; Munster, Andrew Michael; Murray, John Cameron; Nebenzahl, Ben; Nemeth, William; Ng, Patrick Tat Wah; O'Brien, Lorraine; O'Donnell, Barbara Anne; Ogborne, Warren Lambert; O'Grady, Margaret; O'Malley, Terence; Packham, Nicholas Anthony; Pain, Michael Cowper Franklyn; Peak, Howard John; Pearson, Bruce James; Pickworth, John William; Pietzsch, Tibor Thomas; Pigott, Robert Frederick; Pirola, Romano Cesare; Pittar, Mark Rowland; Pokorny-Zsigmond, Akos Jozsef Istvan Zoltan Geza; Poole, Alan Gregory; Power, John Washington; Pritchard, Geoffrey Russell; Pryor, Donald Sidney; Quilty, William Joseph; Quinn, Brian Francis William; Raik, Eva; Re, Bartold John; Redwin, Valerie Margaret; Renwick, Stuart Buckle; Rigney, John Gilbert; Roberts, Harley Stuart; Robinson, Gavin Joseph Brian; Ronai, Ann Kathleen; Ross, Susan Mitchell; Rowe, Peter Brock; Satrapinsky, Zoya; Schiff, Peter; Semple, Bruce; Shearer, Alex Michael; Shuttleworth, Thalia Florence; Sinnathamby, Sivalingam; Skellett, Helen Barbara; Smith, James Robert; Stacey, Bryan

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 25, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1(1)	..	3(1)	..	..	..	..	..	4
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	6	..	6
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(1)	11(10)	1(1)	1(1)	..	1	..	2	18
Diphtheria	..	1	..	..	2(1)	..	..	..	3
Dysentery (Bacillary)	..	..	..	..	2(2)	..	1	..	3
Encephalitis	1(1)	3(2)	..	3(2)	..	..	..	..	7
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	..	..	1(1)	..	..	2
Infective Hepatitis	58(20)	26(15)	6(3)	25(6)	..	..	2	..	117
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	1	..	1	..	2
Leptospirosis	..	..	5	..	..	..	..	..	5
Malaria	..	..	2(2)	..	..	..	..	..	2
Meningococcal Infection	1(1)	2(2)	..	1	1	..	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	1(1)	..	..	..	..	..	..	..	1
Rubella	..	10(5)	..	3	..	..	..	..	13
Salmonella Infection	..	..	..	..	2(2)	..	..	..	2
Scarlet Fever	19(10)	17(16)	2	4(1)	1	1(1)	..	..	44
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	53(1)	..	121	..	174
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	12(8)	18(3)	3(1)	6(3)	10(7)	5(3)	1	..	55
Typhoid Fever	..	..	1(1)	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Arthur Blundell; Stewart, Frederick Harold; Stiel, Mirjam Caroline; Stoksik, Henri Andre; Storey, Brian Gilbert; Tan, Bang Par; Taylor, Jill Argent; Taylor, Roger Bruce; Thew, Roy Farley; Thom, Catherine Theresa; Thong, Ming Chek; Thorpe, Maxwell Elmore Cochrane; Troup, Ann; Turner, Trevor Baxter; Vahl, Stephen Peter; Vincent, Paul Craig; Wane, James Marcus Bennett; Wark, Marian Lynette; Warren, Bruce Albert; Way, Barrie John; Wills, Edward John; Wilson, Peter Hales; Wolfgarten, David Frank Michael; Wong, Rose Swee-Kwong; Wong, Seow Choon; Wright, George Henry; Yap, Yit-Poh; Yu, Chee Keung; Yu, John Samuel.

The following have been issued with a licence in accordance with the provisions of Section 21C of the Act: Briedis, Roberts, M.D., 1930 (Univ. Riga), in respect of the Walisend District Hospital; Garibaldi, Dionisio Italo Guido, M.D., 1949 (Univ. Pavia), in respect of the Dubbo Base Hospital; Nagy, Laszlo, M.D., 1944 (Univ. Budapest), in respect of St. Joseph's Hospital, Auburn; Ropicki, Lydia, M.D., 1948 (Univ. Graz), in respect of the Fairfield District Hospital; Sassi, Armando, M.D., 1944 (Univ. Naples), in respect of the Fairfield District Hospital.

The following has been issued with a Certificate of Regional Registration: Colja, Michael, M.D., 1952 (Univ. Yugoslavia), in respect of the Bogan Gate Region.

Notice.

SEMINARS AT THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY.

ON account of the illness of two members of the panel, the programme has been changed for the seminar to be held on Wednesday, June 3, 1959, at the Women's Hospital, Crown Street, Sydney. The subject for discussion will now be "Surgical Complications of Pregnancy and Labour", the chairman will be Dr. F. Bellingham, and the speakers will be Dr. C. Winston, Dr. R. M. Rawle and Dr. J. Dixon Hughes.

THE CHILDREN'S MEDICAL RESEARCH FOUNDATION OF N.S.W.

THE following is a list of donations to the Children's Medical Research Foundation of N.S.W. received from members of the medical profession in the period March 18 to April 21, 1959. Dr. R. L. Douglas, £5 5s.

Previously acknowledged: £7929 17s. 9d. Total received to date: £7935 2s. 9d.

Nominations and Elections.

THE undermentioned has applied for election to membership of the New South Wales Branch of the British Medical Association:

Hicks, Robert Carey, M.B., B.S., 1957 (Univ. London), Base Squadron, R.A.A.F., Rathmines, New South Wales.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Burchmore, John William, M.B., B.S., 1958 (Univ. Adelaide), 5 Rosedale Avenue, Wattle Park, South Australia.

Lo, Adela Sook Ling, M.B., B.S., 1958 (Univ. Adelaide), c.o. Royal Adelaide Hospital, Adelaide.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Fox, Bruce Spafford Owen, M.B., B.S., 1958 (Univ. Adelaide); Liddell, Robert Victor, M.B., Ch.B., 1923 (Univ. Edinburgh); Altmann, Frank William, M.B., B.S., 1958 (Univ. Adelaide); Moore, Colin Eugene, M.B., B.S., 1958 (Univ. Adelaide); Ancell, Brian Edward John, M.B., B.S., 1957 (Univ. Adelaide).

Deaths.

THE following death has been announced:

BARRON.—George Moncrieff Barron, on May 16, 1959, at Manly, N.S.W.

Diary for the Month.

JUNE 2.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 3.—Western Australian Branch, B.M.A.: Branch Council.
JUNE 3.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 4.—South Australian Branch, B.M.A.: Council Meeting.
JUNE 5.—Queensland Branch, B.M.A.: General Meeting.
JUNE 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 11.—New South Wales Branch, B.M.A.: Public Relations Committee.
JUNE 12.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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